COMPRESSION OF CANCELLOUS BONE*

PRINCIPAL MANIFESTATIONS IN THE HEAD AND NECK OF THE FEMUR

TREATMENT BY CONNECTING DRILL CHANNELS

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DURING the last fifty years a group of pathological changes, involving the head and neck of the femur in the growing child and adolescent, has become increasingly noted.

With the advent of roentgenography the groping progress of differentiation within this group assumed a more vigorous stride. On correlated clinical and radiographic, and in lesser measure histological evidence, this group soon became sharply outlined against other diseases occurring in the hip.

Emerging, as the author traces them, out of the collective vagueness of "interstitial absorption" (B. Bell,[11] 1824) and "nervous coxalgia" (Brodie,[21] 1837, Paget,[81] 1875), this group today comprises the following diseases as accepted entities:

Osteochondritis dissecans of the head of the femur, the several forms of coxa vara, the Waldenstroem-Legg-Calvé-Perthes' disease, the slowly slipping, and the acutely slipped, upper femoral epiphysis and the deforming process subsequent to congenital dislocation of the hip.

Concurrently with the mentioned manifestations in the femoral head, changes were observed in the acetabulum, or rather the bones that form this and the pelvis, and "osteochondritis of the acetabulum" (Fromme,[45] 1920) and "acetabular Perthes' disease" (Gaugele,[46] 1931), "ischio-pubic osteochondritis" (Van Neck,[110] 1924), "osteochondritis of the symphysis" (Burman,[24] 1937) and

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"adolescent sacroiliac syndrome" (Rogers,[95] 1935) were added to the list.

Due to similarities of features with the separation of the upper femoral epiphysis, but mostly due to obscurity of nature, the juvenile fractures of the femoral neck were drawn into this group under investigation.

The author believes that all these conditions with some others occurring in different parts of the body, and at present not thought related, are based on one single pathogenetic element, and he shall endeavor to evolve a concept of the same which, if given the key position it merits, shall clear the prevailing confusion and shall facilitate the understanding of the grosser features of the whole group.

Further, a therapeutic principle arising out of this concept will be outlined and illustrated.

The prevailing opinion, as expressed in current literature, allocates the origins of our knowledge of this group, with the exception of osteochondritis dissecans, at the earliest to E. Mueller[79] (1888), or Fiorani[42] (1881), and that of the most puzzling of them to Waldenstroem[113] (1909) and Legg[68] (1910).*

EARLY RESEARCH

Bibliographic investigations of the author, however, led him to uncover material that demonstrates that the recognition and accurate description of some of the important forms of these diseases date back as far as 1785 and earlier, and that most of these conditions were subsequently often encountered and described, and that B. Bell[11] in 1824, and Ollier[80] in 1881 formulated strikingly correct concepts concerning the underlying process.

These concepts, especially that of Bell, were readily accepted by their contemporaries, and were progressing in what the author believes to be the right direction, when they were suddenly sidetracked by the sensational appearance of coxa vara in 1888.

The products of this medical research, which spread over almost a century, are little if at all known. Their noteworthy contents deserve detailed analysis, but in this paper they will be discussed

* Omitting at this time such precursors as Sindig-Larsen, Koehler, G. Schmidt, Frangenheim, etc., and in general writers on juvenile arthritis deformans from Maydl to Perthes.

only in a measure necessary to render an account of the concepts they represent, and to substantiate the claims made before.

During this period under consideration scientific research consisted mainly of correlating clinical observations with the gross pathological specimen. These specimens in the field of bone pathology were furnished in England in substantial numbers by poorhouses and community hospitals that sheltered and cared for the aged and the pauper.

It is in the nature of such a group of patients that, if some among them had suffered earlier in life from any of the conditions of the hip we are concerned with, their autopsy decades later would show far advanced or end stages of these diseases. This latter circumstance introduced an additional confusing element into this complicated matter, namely, that of the intracapsular fracture of the neck of the femur.

It is easy to see that a hip which underwent in childhood or adolescence some of the diseases enumerated, may present at advanced age a strikingly similar picture to that of an intracapsular fracture of the neck of the femur, that by chance resulted in bony union with more or less shortening of the neck.

It was, however, from this overlapping of the two groups that a slow clarification began of the problems involved; for it became increasingly evident that intracapsular fractures of the neck of the femur treated by contemporary methods seldom, if ever, progressed to bony union (Cooper,[29] 1819) and consequently those specimens that showed what was claimed to be bony union of fractured femoral necks, must have been the product of some "morbid action" (B. Bell,[11] 1824) or "altered state" (Cooper,[30] 1825).

Cooper and his collaborators collected an impressive number of cases of fractured femoral necks that were observed through varying lengths of time from accident to autopsy, and have gradually driven home, over English (Earle, etc.) and French (Dupuytren, Desault, Roux, etc.) opposition the tenet of nonunion that was to hold sway until Whitman[121] (1902).

The more this became accepted, the more critical attention became concentrated upon those specimens which were offered as examples of bony union. The few older specimens available for inspection such as that of Trioen[108] (1743), were declared erroneously judged, and based on a large amount of material, B. Bell[11] formu-

lated his conception of "Interstitial Absorption of the Neck of the Thigh-Bone," which after years of oral teaching was published in 1824.

According to this concept: "In an advanced period of life, some-

FIG. 1. Bell's illustration of "interstitial absorption of the neck of the thigh-bone" 1824.

thing resembling a yielding or bending of the neck of the [thigh] bone may be perceived. This effect does not proceed from any softening or alteration in the intimate texture of the bone but arises in consequence of the absorption and disappearance of a portion of its entire substance. Sometimes, more than two-thirds of the neck of the bone disappearing, so that the head of the bone is as if it were forced in the direction of its axis towards the base of the trochanter major. In some rare cases (Fig. 1) combined with the shortening of cervix femoris, there is also a flattening of the head of the bone. Superadded [to these changes] . . . frequently the lower part of the neck of the bone [is] seemingly incased in a sheath of osseous matter. It is these cases (Trioen) [Fig. 2] that are apt to be confounded with fractures of the neck of the thigh-bone.

"This affection, incidental to the neck of the thigh-bone, I [Bell] termed interstitial absorption.

"This is a disease of frequent occurrence, but nevertheless has been almost overlooked by systematic writers, or confounded by them with fracture, dislocation, mollities ossium, and scrofulous affections of the hip-joint.

FIG. 2. Supposed bony union of an intracapsular fracture of the neck of the femur, Trioen, 1743. Interpreted as "interstitial absorption" by Bell.

"It rarely occurs in conjunction with rickets—it is a purely local affection, and does not involve much constitutional irritation—and differs from inflammatory and scrofulous affections occurring in the hip-joint."

When first formulated this concept of Bell's seems to have been more or less a corollary to the much discussed topic of the day, to Cooper's thesis of the prevalence of nonunion of fractured femoral

necks. It gave support to this by finding an explanation of those cases that were offered as instances of bony union.

In retrospect it may be said that Bell in his first publication

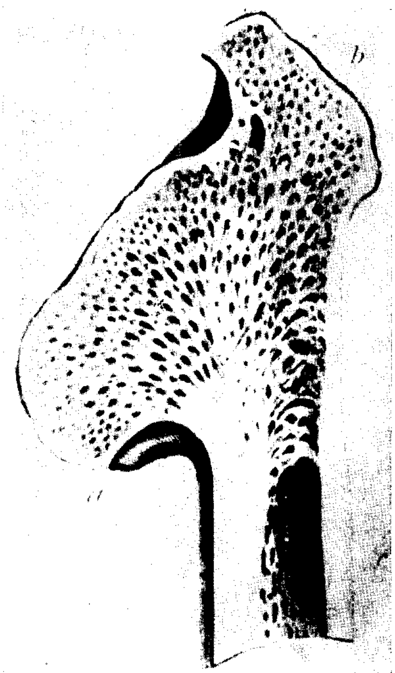


FIG. 3. Cooper's illustration of an extreme case of interstitial absorption, 1825.

described essentially, as the author sees it, the clinical picture of the incomplete or fissure fracture of the neck of the femur that progresses to absorption of the neck and subsequent deformity; a clinical picture which even today is by far not as well known or appreciated as it deserves in spite of the fact that it was introduced into American literature by Hamilton[51] in 1860.

Soon, however, the material of Bell and his followers passed beyond the narrow limits of this one entity, and four years after his first publication, Bell had to enlarge the frame of his concept.

The changes that he believed to be incidental to old age in the meantime, were observed at autopsies of persons other than aged.

"I [Bell] have met with cases since the publication of my memoire on this disease, in which interstitial absorption affected the neck of the thigh-bone of one side in persons of thirteen, thirty, and forty

years of age—and my friend Dr. Knox[62] has related a most interesting and curious example of this affection occurring in the neck of the thigh-bone of a child three years of age."

Further observations of Bell have made him not only extend the age limit of this "interstitial absorption," but also to recognize that the same process may occur in other parts of the skeleton than the neck of the femur, and he intimated the entity which, later outlined if somewhat vaguely by Stanley,[104] Russell[96] and McNamara,[74] eventually became known as Kuemmel's disease of the spine.

One glance at the illustrations given by Bell or Cooper (Fig. 3) or at the case history and autopsy report of Knox is convincing enough to show that what they had before them were instances of the disease or diseases that are the subject of this paper, diseases which today more than a century after these authors' first efforts still lack a satisfactory explanation.

But even they were not the first ones to observe, describe and illustrate these conditions and to formulate an opinion about their nature. Following a reference given by Bell, the author has come upon a work of Giovanni Baptista Palletta[82] who in a monograph in 1785 entitled "On Congenital Limping" (De Claudicatio Congenita), has given a remarkable clinical and pathological description of certain forms of coxa vara and some related conditions with little admixture of other confusing matter.

The work that antedates E. Mueller[79] by more than a hundred years is practically unknown or grossly misinterpreted. Writing ninety-six years after Palletta, his compatriot Fiorani,[42] for whom credit was lately claimed for the description of coxa vara in preference to Mueller, makes no mention of him.

The author considers this work of Palletta as the earliest classic on the conditions dealt with in this paper. It may well be a satisfactory starting point as Palletta, in order to avoid the accusation of indulging in "inane speculations" confirms his statements by autopsy reports and gives an engraved illustration of his most important case. (Fig. 4.) Palletta hands the concept of coxa vara chronologically still farther back to Morgagni, his one-time teacher, whom he credits with the establishment of the fact that shortness and transverse position of the femoral neck may reduce the total length of the limb and cause limping. He gives full description of the autopsy reports of Morgagni's cases, then shows that these findings in the aged, recorded by Morgagni, may also be found in

young persons. He explains these cases due to the absence of manifest pathological changes as congenital malformations and creates the entity of "congenital limping," the first recorder of which he professes to be.

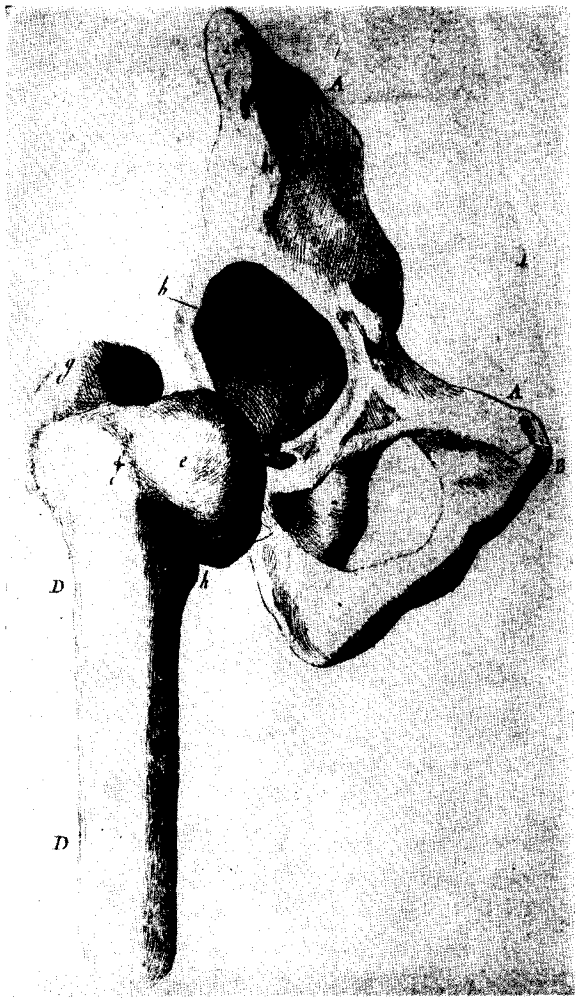


FIG. 4. Palletta's specimen of coxa vara, 1785.

According to Palletta these changes may assume the following characteristics: (1) Shortness to total consumption of the femoral neck; (2) decrease of the neck-shaft angle to right angle and less; (3) subsequent to these changes, lowering of the head below the level of the apex of the greater trochanter and the shaft; (4) en-

largement of the head and also of the acetabulum; (5) oval or flattened deformity of the head with occasional mushrooming of the head over the neck; (6) torsion of the neck anteriorly relative to the shaft; and (7) torsion of the head backward on and relative to the neck.

Aside from these specific findings he emphasizes the generally normal appearance of the joint, the normal aspect of the synovial fluid, the absence of gross alterations of bone and cartilage, no indication of injury; in short, no pathologic findings except the deformity.

The clinical picture Palletta gives is equally as clear, mentioning: (1) Shortening of the limb; (2) flattened buttocks with up and outward curving of the gluteal fold; (3) high stand and lateral prominence of the greater trochanter; (4) absence of marked weakness or wasting of the limb; (5) absence of pain; (6) marked freedom of motion in the joint; (7) emphasizes that it indicates no affection of general health, no lessening of pelvic space (caliber) and no interference with normal parturition.

Claiming credit for Palletta, it must be stated that similar findings were made before him and specimens were kept as curiosities in many collections. Palletta himself mentions the Hovianus Collection of Andreas Bonn[15] with several well described specimens. A study, however, of Palletta's monograph will doubtless prove that he was the first who clearly correlated the clinical features in the living with autopsy findings, and consciously outlined an entity which had to be rediscovered laboriously after his own work was long forgotten.

This oblivion is a bibliographic curiosity. Thirty-five years after his work on congenital limping Palletta, in 1820, published observations on congenital dislocations of the hip antedating Dupuytren[36] by six years. Soon the commentators with rare exceptions (Ammon[2]), misled perhaps by the title of congenital limping, confused the earlier work of Palletta with his later one. Finally, as the author found it, Carnochan,[28] in 1850, prominently featured and firmly established this error by extensive but misinterpreted quotations.

Palletta's contemporaries did not fail to recognize the importance of his work. It was soon translated into German (Tabor, Heidelberg, 1791). In Holland it was reprinted in its original Latin and Palletta's most important specimen with its case history was incorporated into the monumental work of the great anatomist-atlas-makers,

Eduard and Gerard Sandifort,[98] in 1793–1835. It was through this atlas that the case came to the notice of Bell who reprinted it in full and interpreted it as juvenile occurrence of interstitial absorption.

For the Sandifort atlas new drawings were made of Palletta's

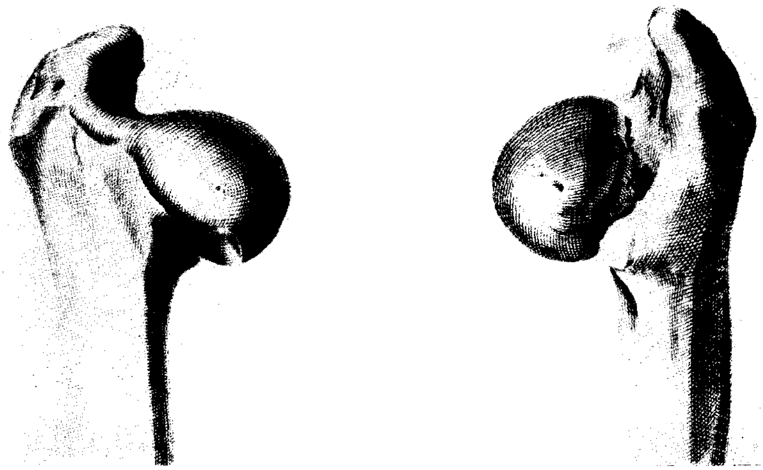


FIG. 5. Palletta's specimen as reproduced in the Sandifort Atlas, 1793–1835.

specimen. These drawings (for this time there are both an anterior and posterior view) are far superior to Palletta's original engravings, and make it possible to identify Palletta's case as that of the late stage of slipping femoral epiphysis. (Fig. 5.)

The two sets of illustrations, while similar enough, differ in detail quite a bit; but it was possible to establish their identity through the statements in the atlas, the works of Paletta, Bell, and E. C. A. Sandifort,[91] the son and grandson, respectively, of the atlas makers, who in 1834 in a monograph on "Congenital Pathological Conditions of the Hip," restates and elaborates Palletta's concept.

These authors, the Sandiforts and Bell, clearly recognized the portent of Palletta's findings. In Holland, through the efforts of the Sandiforts, this disease became well known and was kept apart from the congenital dislocation of the hip. Bell sensed its relationship to interstitial absorption observed in elderly persons. In Germany, thanks to Ammon's[2] faithful following of E. C. A. Sandifort's monograph, this disease became clearly outlined and illustrated (1842). Thus, the early entry of this clinical picture was equally as forceful as its reappearance one hundred years later as coxa vara at the hand of E. Mueller.[79]

In view of this widespread recognition it is strange that all these efforts became forgotten. The more so as in England, following directly upon Bell's work, a group of investigators brought the knowledge of these conditions to a marked degree of development.

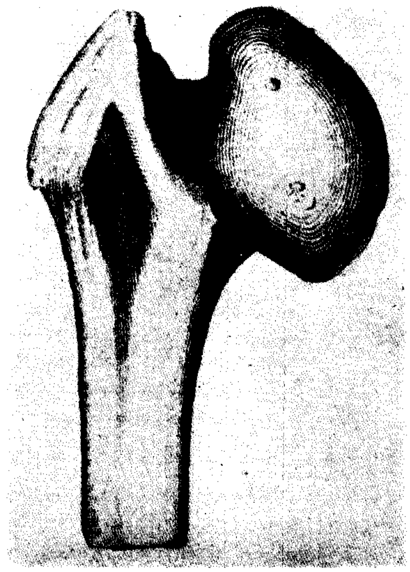


FIG. 6. Gulliver's illustration of "interstitial absorption" in a nineteen year old recruit, 1836.

The first among them was Gulliver[48,49] who in 1836 in two publications stressed the need of clarifying "how far interstitial absorption and shortening of the neck of the femur may be the effect of disease at an early period of life," described several such instances and illustrated one occurring in a "stout recruit" at the age of nineteen. (Fig. 6.)

Knox,[63] in 1843, reports the autopsy findings of yet another juvenile case of shortened femoral neck, and recalling his case referred to by Bell, brings forth the congenital theory of the deformity as his own, apparently not knowing of Palletta.

Canton, in 1848, in a paper,[26] and in 1855 in a monograph[27] consolidates the advances made by Bell, Gulliver, and Knox, and adds to it the clear differentiation of interstitial absorption from tuberculosis of the hip, the "morbus coxarius" of his day—"this latter complaint [morbus coxarius] leading not infrequently to total disorganization of the joint and ankylosis, or terminating in death;

the former [interstitial absorption] being an affection unconnected except incidentally with the strumous diathesis, and ending in loss of the cervix femoris without producing any constitutional disturbance, without the establishment of ulcerative absorption, the formation of matter, involving to a variable extent the head of the bone and the acetabulum, but not affecting eventually to any degree the range of motion."

Proceeding beyond the clinical recording, Canton arrives at a concept which in the opinion of the author is essentially correct: "Where the head and the neck of the femur . . . suffer concussion only . . . where indeed these parts are submitted to a momentary compressing force, passing between the trochanter and the opposite point of resistance, the acetabulum, the same change [interstitial absorption] is liable to take place."

All this Canton rounds out with the recognition that injury to the ligamentum teres may impair the circulation in the femoral head. This is a point on which, however, the youngest Sandifort and Palletta himself have made abundant observations.

The writings of Gulliver and Canton picture the positive progress of knowledge of the shortening of the femoral neck and the lessening of its angle. Reading them, one wonders why, in spite of these observations and this intimate acquaintance with these deformities, orthopedic surgery had to wait for E. Mueller to introduce the concept of coxa vara. The answer to this puzzle is amply given in the works of Gulliver and Canton, especially the latter.

As was mentioned before, the interest of Cooper and Bell culminated in establishing the doctrine of nonunion of the fractures of the femoral neck. They bent to this purpose all their observations. Excluding the possibility of union in cases of intracapsular fractures of the neck of the femur, they used the concept of interstitial absorption to explain the numerous autopsy findings of shortened necks and sessile heads. To explain away the cases with reduced angle but maintained length of the neck, they resorted to the arbitrary assumption of a quasiphysiological occurrence of interstitial absorption, an "idiopathic affection" that was supposedly a manifestation of senescence. By the adoption of this view they have blocked the path to the recognition of coxa vara, on which they so promisingly started.

That this crude outline is in a good measure true to the actual happenings, one may easily gather from Canton.

Canton more than Gulliver before him, is disinclined to accept the lessening of the angle of the femoral neck as part of senile involution but, deferring before the authority of Cooper, smothers his own doubts under an imposing array of quotations from contemporary literature.

He lines up Knox,[61] Harrison,[52] Cruveilhier,[32] Wilson,[122] Elkington,[37] Cooper,[30] Erichsen,[38] etc., teachers, lecturers, authors all; they testify that the lessening of the angle of the femoral neck is a sign of old age. Thus, this learnedly supported error of first magnitude became firmly established in England and also in France. It was rather late when Humphry,[53] in 1889, adopting the stand made by Lane[67] (1886), in a large and thorough anatomical study corrected this misconception, for in the meantime Mueller,[79] Hoffmeister and Kocher made the medical world acquainted with the coxa vara and no notice was taken of the fact that Humphry clearly described the coxa valga.

The concept of interstitial absorption, in particular the knowledge of its juvenile occurrence was kept alive in England with publications by Fayrer[40] in 1862, Quain[87] in 1884, and Jones[55,56] in 1882 and 1887. Of this latter group of writers Quain in his textbook restates the concept in great detail and by adding his own clear and correct opinion concerning the pathological process, marks the high point of development of Bell's idea. It is of interest that Quain gives the photographic illustration of Gulliver's specimen preserved in the Army Medical Museum, which bears out Gulliver's description more strikingly than the engraved sketch accompanying his article forty-eight years earlier. (Figs. 7, 8 and 9.) Jones' textbook closes English publications, as far as the author could ascertain, on this topic in 1887, the year before Mueller announced the coxa vara.

The period of Cooper produced another clinical and pathological picture, the malum senile coxae. The origins of this entity became somewhat obscured by the vicissitudes it underwent soon after it came into being. The first and thorough description was given by R. W. Smith[103] (1834). This entity, which in the last hundred years has grown into the problem of arthritis deformans is, in the opinion of the author, intimately connected and fundamentally identical with all the other conditions that are the subject of this paper.

Smith considered trauma the causing factor, but obligingly deferred before Stanley[104] who suggested "chronic rheumatic" influences; and it was under the name of "chronic rheumatic

arthritis" that Adams[1] incorporated it in the Cyclopaedia of Anatomy and Physiology in 1836–1839. The designation malum senile coxae, however, survived probably due to the fact that

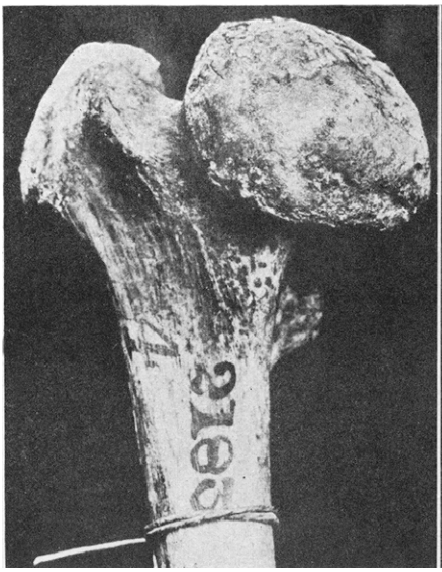


FIG. 7. Gulliver's specimen photographed forty-eight years later by Quain, 1884.

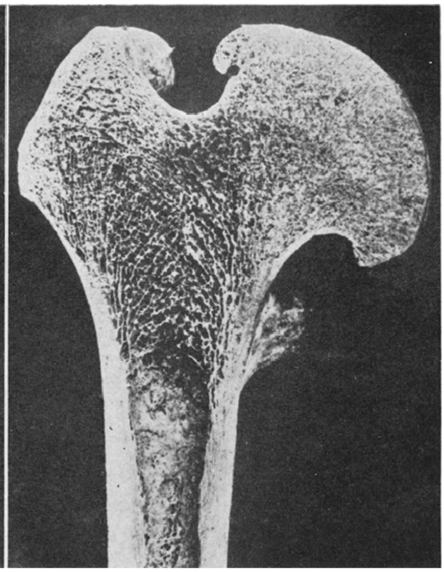


FIG. 8. Same specimen as in Figure 6, cross section, Quain, 1884.

Wernher[117] enthusiastically transplanted the concept to Germany under this name. There at the hands of Virchow and Ziegler[125] the topic received thorough investigation. Ziegler's work in 1877 on subchondral changes, laid the modern foundations of arthritis deformans leading eventually to the differentiation of its juvenile form by repetition of the path evolved by B. Bell and his followers in England. It is of interest to note that Ziegler's findings were described and illustrated by Canton[26] in detail in 1848.

France has actively participated in the investigation and discussion of topics brought forward by Cooper and his contemporaries. A great deal of material on these deformities was demonstrated and published mostly under the name of "arthrite sèche" somewhat confusingly, as this term eventually became identified with Adams' "chronic rheumatic arthritis" (Quain, R., Dictionary of Medicine, 1895). No detailed account of this material will be given in this paper, particularly because a publication of Ollier,[80] in 1881, dwarfs them all by its importance.

In his article "Juxta-Epiphyseal Sprain" (De l'entorse juxta-épiphysaire . . .) Ollier outlines a concept of obscure injuries caused by forced movements, in joints of children. He envisions

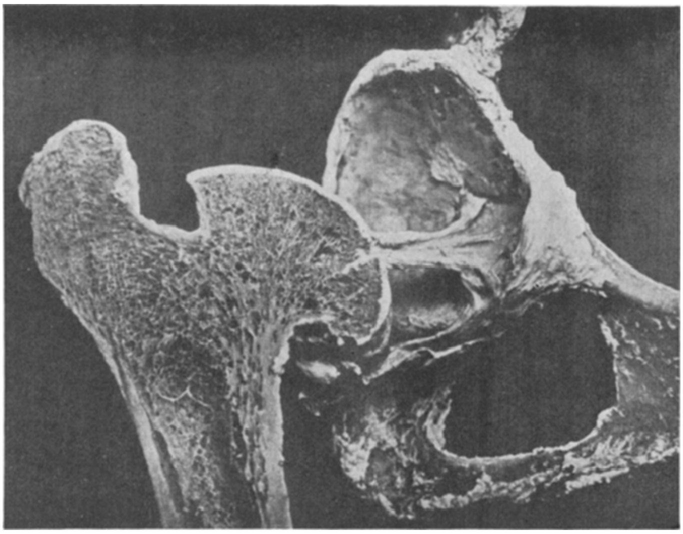


FIG. 9. Specimen of another case of Gulliver's. Quain, 1884.

and experimentally produces (Fig. 10) "intimate lesions of the juxta-epiphyseal spongiosa" and reasoning "on its consequences on the nutrition of the bone," he correctly concludes that "these injuries are the first degree of epiphyseal separation." In this short review no adequate justice can be given to this work of Ollier, which shows the problem in many of its details with great clarity.

These bibliographical findings proved to the author that the knowledge of these various deformities occurring in the hip was more or less thoroughly familiar considerably prior to Mueller. The question remained whether the elusive clinical picture that accompanies the course of these deformities in the making, escaped the observant clinicians of this period.

There is ample evidence to show that they were indeed conscious that under the blanketing manifestations of tuberculous hip disease (which under other names they well knew) there hides at times a more diaphanous symptom complex.

Brodie,[21] in 1837, speaks of hysterical manifestations in the hips "even of young males." Paget,[81] in 1875, speaks of nervous mimicry. With the development of the concept of hysteria and the differentiation of Charcot's disease the discussion centers on the hip joint, and

coxalgia (Fricke[44] 1833), nervous coxalgia, pseudocoxalgia become intensely debated topics. The voluminous literature that resulted cannot be given space in this article, though it is of bibliographic

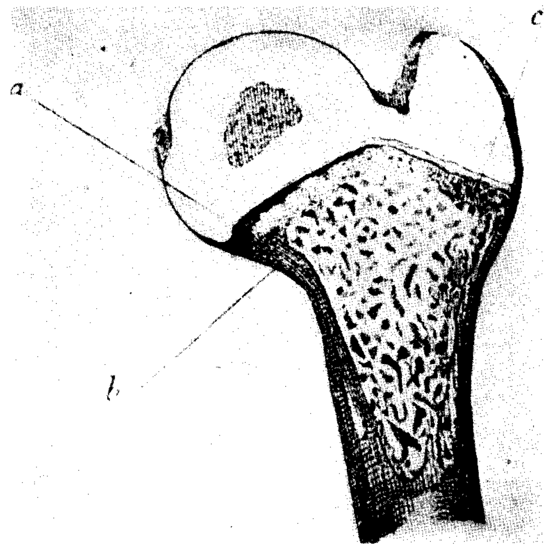


FIG. 10. Beginnings of epiphyseal separation experimentally produced by Ollier, 1881. *a*, beginning separation; *b*, periosteum; *c*, crushed bone tissue.

interest to follow Wernher,[118] Minich,[77] Bauer,[9] Shaffer,[99] and others as they segregate the symptoms that, as we know today, accompany the slipping of the femoral epiphysis and the course of the Waldenstroem-Legg-Calvé-Perthes' disease.

Gibney's[47] construction of the clinical picture of the "neurosis of the hip" without knowledge of the underlying process cannot fail to impress any clinician. He speaks of "age of occurrence around or before puberty," of "difficulty of differentiation from true hip disease," "quick fatigue and muscular spasm," "recurrence of symptoms after remissions," "peculiarity of gait indicative of pain or fear, otherwise difficult to describe," and occasional "prominence of the affected hip."

The tide of discussion abruptly subsided with the demonstration of other than nervous bases of these "hip-joint neuroses." This topic reached greatest development and survived the longest in France where Calvé[25] himself described the disease named with others after him, as a "particular form of pseudocoxalgia."

A survey of the American literature of this period also led to interesting findings.*

Richardson,[90] in 1860, twenty-eight years before Mueller,

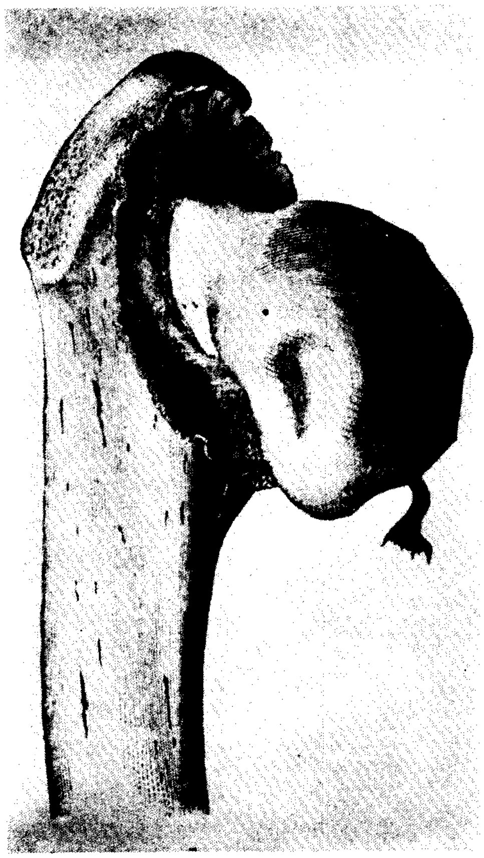


FIG. 11. Richardson's specimen of coxa vara, 1860. Imprint on the head, possibly primary focus.

describes and illustrates coxa vara in a twenty-five to thirty year old male. By the bilaterality of the deformity he excludes healed fracture of the neck, describes the head "enlarged, spread-out, and rendered irregular at its junction with the neck by deposits of little nodules of new bone." (Fig. 11.)

Monks,[78] in 1886, describes "a case of unusual deformity of both hipjoints" that developed in the course of two years in a sixteen year old boy. The lucid clinical analysis together with his

* Known to R. Whitman.[120]

line drawings (Fig. 12) support the assumption of bilateral slipping epiphysis that progressed to coxa vara deformity and enlargement of the head. Monks himself is "forced" to conclude that the case

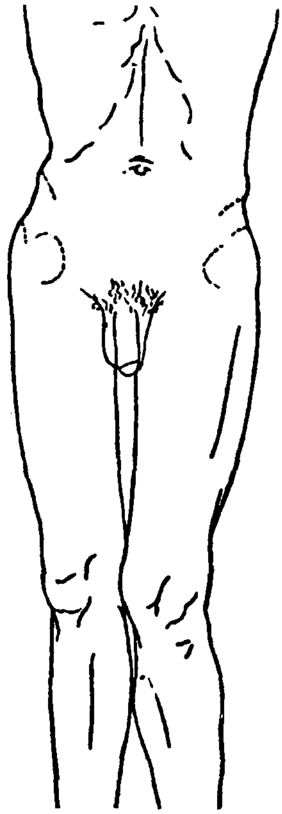


FIG. 12. Monk's illustration of coxa vara, 1886. Note *dotted line contour of* upper end of femurs.

is juvenile occurrence of arthritis deformans, and thus is the first one to consider this possibility and to use this term with equally as much justification as those who proposed it later.

The concept of Bell's interstitial absorption also found its way to America. Beside Hamilton's broad view of the topic in 1884, Vance,[111] taking his start from Quain, gives a thorough résumé of the concept and illustrates it by a case of his own in 1890.

The description of coxa vara by E. Mueller in 1888 may well be considered the beginning of the modern period of orientation on this and related subjects. The huge mass of publications that resulted

will at the present not be discussed as they are more or less well known.

The author wishes to make one exception concerning an article

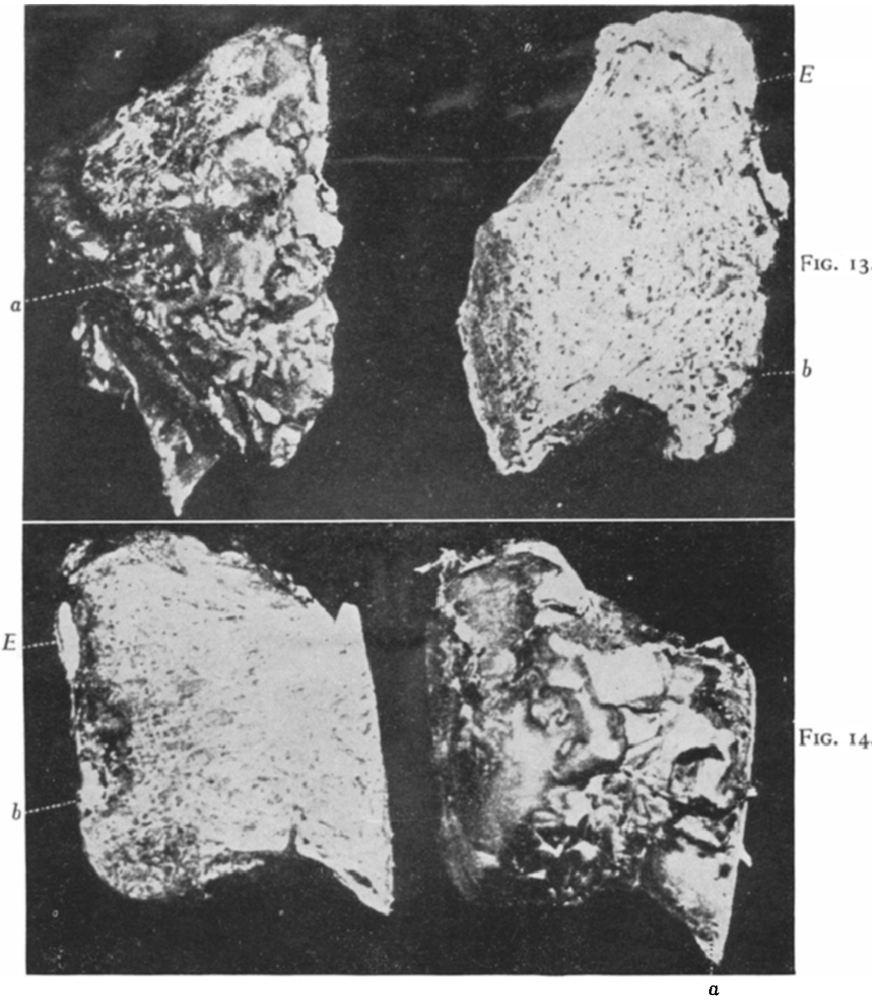


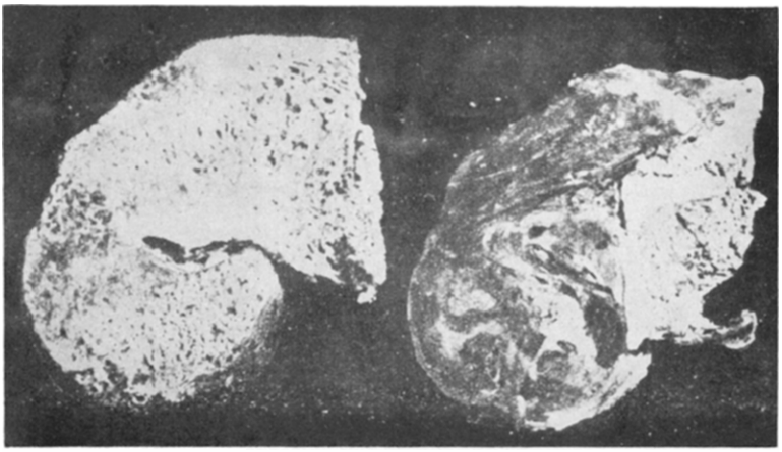
FIG. 13. Maydl's specimen (1897) of what later became known as Legg-Perthes' disease. *a*, posterior view; *b*, cross section; *E*, remnants of the femoral head.

FIG. 14. Another specimen of Maydl's, of the same condition as in Figure 13. *a*, anterior view; *b*, cross section; *E*, remnant of the femoral head.

of Maydl,[72] the importance of which apparently escaped most reviewers.

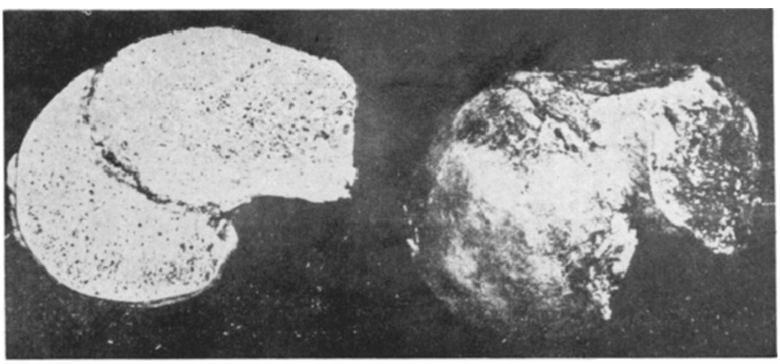
In 1897, Maydl published the photographs of four resected femoral heads that are, as the author could ascertain, the first

well recognizable specimens of Waldenstroem-Legg-Calvé-Perthes' disease (Figs. 13 and 14) and slipping upper femoral epiphyses. (Figs. 15 and 16.)



b a

FIG. 15. Maydl's specimen of slipped femoral epiphysis. *a*, anterior view; *b*, cross section.



c d

FIG. 16. Another specimen of Maydl's, of the same condition as in Figure 15. *d*, anterior view; *c*, cross section.

In addition, Maydl reveals that the deforming process following traumatic dislocation of the hip was known to him as the traumatic variety of arthritis deformans (Zesas). To this and the original idiopathic (senile) form he proposes to add, on the basis of his four cases, the juvenile form of arthritis deformans which he wishes to separate from the "recently described" coxa vara. Thus, it was Maydl who first conceived this form and used the term in Europe and not Perthes, as is at times believed.

The literature prior to 1888 with the addition of Maydl's publication shows to the satisfaction of the author that the conditions to be discussed in this paper were well known prior to the modern period of research, and that this latter period produced until recently the rediscovery by *x*-ray of the same conditions that were so accurately described in the course of the century before radiography.

COMPRESSION OF CANCELLOUS BONE

After more intimate observation the diseases enumerated and some others occurring in different parts of the skeleton began to be considered interrelated. Notable attempts at systematic grouping were made by Burckhardt[23] who gathered them around the central entity of arthritis deformans, following the course of emergence of these diseases in the German literature, and by King[59] who aligned them as "rarefying conditions of bone" reminiscent perhaps of Bell's approach.

The connecting link of these conditions is, as generally accepted, the aseptic necrosis of the bone involved. The knowledge of aseptic necrosis of bone, the quiet necrosis of Teale[107] and Paget,[81] has attained exact clarification by Axhausen,[4,5,6] Cordes,[31] etc., in Germany, and Phemister,[85] in America. Through the efforts of these authors and others, this process is well known in many of its aspects with the notable exception of the question of how the necrosis originates.

Of all the proposed explanations the "infarct theory" of Axhausen is the most prominent. This theory is based on the observation that the necrotic areas often show on cross section the wedge shape characteristic of infarcts. These infarcts, according to Axhausen, are caused by "bland emboli" that do not produce suppuration and thus leave the necrosis aseptic.

This theory, in spite of the fact that bacterial and air-emboli actually were demonstrated as causes of bone infarcts, encountered scepticism, even among its early supporters. Thus far, however, no other explanation was found plausible enough to be accepted in its place.

The author—as he believes—has found an explanation that solves the question of the underlying cause of aseptic bone necrosis and clarifies the relationship of its numerous clinical manifestations.

The characteristic wedge-shaped areas of aseptically necrotic bone may be produced by a mechanism other than infarction,

namely, by compression of a spherically-shaped portion of cancellous bone. The results of compression applied to a spherically-shaped body are illustrated in the example of a bruised apple in Figures 17 to 21.

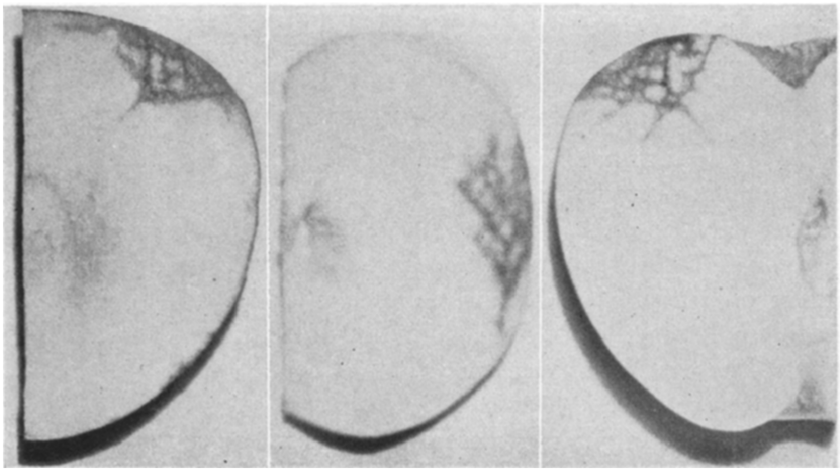


FIG. 17. FIG. 18. FIG. 19.

FIG. 17. Typical compression wedge in a bruised apple.
FIG. 18. Double wedge following double impact.
FIG. 19. Compression wedge showing bisecting compression planes.

They demonstrate the following: (1) If subjected to compression, a spherically-shaped body will suffer damage in an area that is wedge-shaped on cross section. (2) In three-dimensional actuality this area corresponds to a cone with its base at the site of application of force. (3) The compression does not damage the whole substance of the cone, but is most effective on the jacket of it. (4) Within its compression pattern, secondary component wedges are demonstrable. (5) Each impact produces its own wedge. (6) The wedge itself is produced by bisecting compression planes, inside of which the substance may be undamaged. (7) Changes in the curvature of the surface and the speed of force alters the shape of the compression pattern, and if the change occurs during the insult, combination patterns result and patterns may superimpose one another.

By correlating these observations with the fact, the significance of which escaped notice, that all the diseases under consideration occur in cancellous bone and at such places where the bone presents a more or less spherical surface, the author arrived at the concept of compression of cancellous bone which, he is convinced, is the

sole underlying cause of all these diseases, and may well be considered an entity because, while manifest in a variety of clinical pictures, it presents at all times the same pathological features.

If cancellous bone is subjected to force which is greater than its

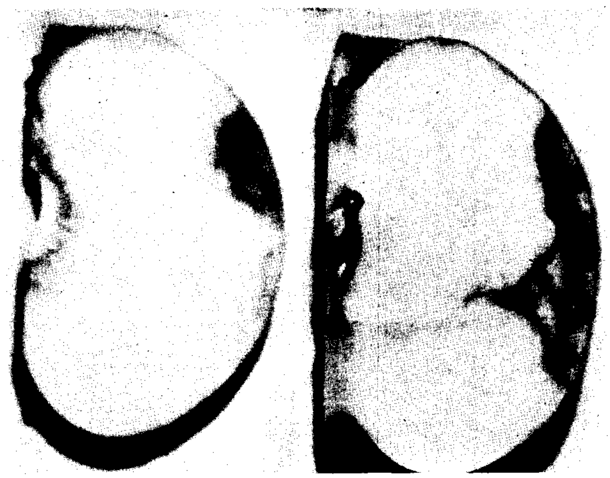


FIG. 20. FIG. 21.

FIG. 20. Compression focus of rounded contour produced by slow pressure.

FIG. 21. Compression figure by sharp impact followed by rolling. The wedge shows maximum compression on the outer borders and undamaged areas between secondary compression planes.

maximum of structural strength, its architecture will collapse. This collapse obeys the laws of mechanics, and the minute girders of the cancellous structure break at places predetermined by these same laws. The broken mass of trabeculae interrupt the flow of blood and causes necrosis of certain, also predetermined segments of bone.

As illustrated, in the case of a spherically-shaped segment the place of collapse is the jacket of a cone, and this whole cone is rendered necrotic, not by embolism of a vessel entering the apex of the cone as presumed by Axhausen, but by compression of all and any vessel entering the cone on its entire jacket.

The necrosis thus mechanically produced is aseptic.

Mechanical factors underlying the occurrence of aseptic bone necrosis were and are considered by many writers. Axhausen[4] himself, prior to his adoption of the infarct theory, believed in traumatic etiology. Walter,[114,115] formerly a supporter of the embolic hypothesis lately also accepted the traumatic view. Mau[71] in his ingenious experiments laid new foundations for the mechanical

theory. Cordes[31] in his remarkable studies effectively advanced the knowledge of mechanics in question. In his lucid invocation and illustration of the laws of mechanics in osteochondritis dissecans Roesner[93,94] came near to the view of the author. He failed, however, to realize the identity of the far-flung manifestations and was misled hereby to his forced and untenable explanation of the infarct pattern which he adopted later.

The parts of the skeleton that harbor these diseases contain besides cancellous bone, also cartilage, in particular in childhood and adolescence. Cartilage is in its physical properties fundamentally different from cancellous bone and reacts to compression differently. It was fair to assume that being more elastic the cartilage is less "vulnerable" to physical injuries, especially to compression. Opinion, however, to the contrary was and is occasionally expressed and the author, therefore, endeavored to verify the behavior under compression of these two component elements in question. Femoral heads of rabbits were thoroughly crushed and examined. Under the microscope it was found that while the cancellous bone was transformed into an unrecognizable mass of débris, the minute particles of cartilage retained their normal appearance. The assumption, therefore, that it is the cancellous bone that suffers most of the damage by compression and that cartilaginous parts escape more or less unharmed, seemed justified.

In applying these premises to the compression of the femoral head at different stages of development, that is, at different age periods, it became obvious that the different clinical forms observed in children and adolescents are determined solely by the ratio or relative amount of cartilage and bone in the femoral head at the time of injury.

INDIVIDUAL MANIFESTATIONS

A. In the Femoral Head. A more or less fully developed femoral head covered by the normal thickness of articular cartilage will respond to compression as illustrated in the diagram in Figure 22. This the author considers the mechanism of origin of the osteochondritis dissecans, the prototype of all lesions, as it represents compression of cancellous bone in its least complicated form. This mechanism explains the occurrence of this disease only in curved segments of cancellous bone, and the presence of undamaged bone within the free bodies. The secondary compression planes answer

the question rightly raised by King[59] concerning multiple free bodies associated with a single bed.

The femoral head that harbors in a young child a smaller or

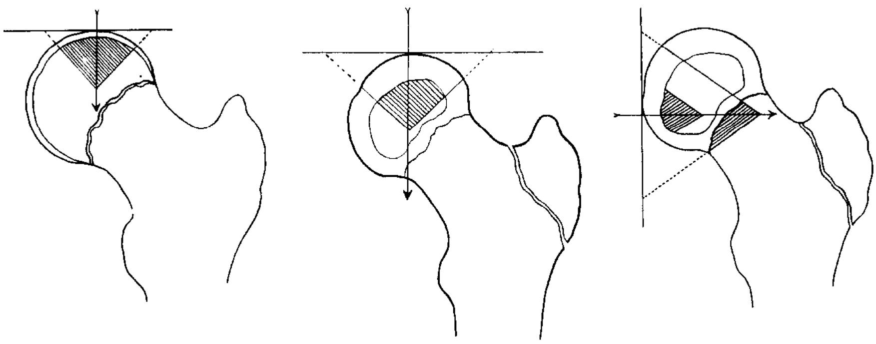


FIG. 22. FIG. 23. FIG. 24.

FIG. 22. Mechanism of origin of osteochondritis dissecans.

FIG. 23. Mechanism of origin of Waldenstroem-Legg-Calvé-Perthes' disease.

FIG. 24. Mechanism of origin of slipping femoral epiphyses. Note double foci.

larger osseous center beneath a thick cartilage covering responds to compression as illustrated in the diagram in Figure 23. The cartilage itself escapes the damage of compression which becomes effective only in the bony center. Observation of his cases over prolonged periods revealed to the author that while the cartilage escaped harm, the vessels traversing it on their way to the ossification center also may suffer from compression. The effect of this develops later and brings forth in the subsequent osseous growth of the head the typical compression crater at the site where it was kept latent previously by the cartilage, as will be illustrated in the case reports. This diagram illustrates the mechanism of origin of the Waldenstroem-Legg-Calvé-Perthes' disease, which will be recognized as essentially a subchondral osteochondritis dissecans, occurring under the thick cartilage of the femoral head. In support of this mentioned relationship of the two diseases the author wishes to point out the parallel of occurrences in the capitellum of the humerus. In the fully developed capitellum osteochondritis dissecans occurs; while in earlier stages in the presence of a small ossification center and thick cartilage covering, the disease named after Panner[83] develops, which is identical with the Waldenstroem-Legg-Calvé-Perthes' disease, differing from it only insofar as it is determined by the absence of weight-bearing.

A femoral head somewhere between the two stages illustrated, will respond to compression according to the diagram in Figure 24. Articular and epiphyseal cartilage escape compression, which is

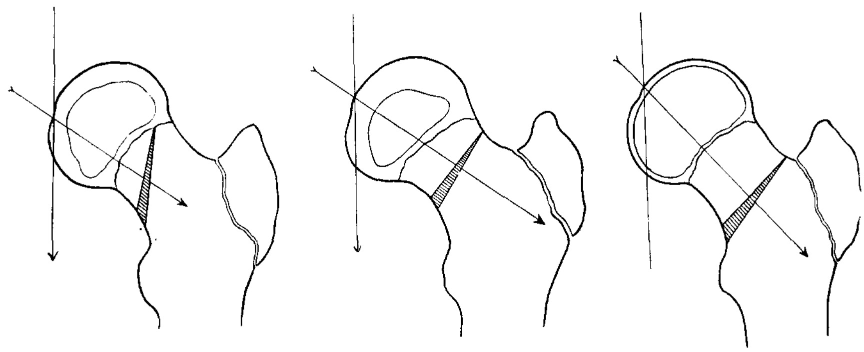


FIG. 25. FIG. 26. FIG. 27.

FIG. 25. Compression wedge close below the head, crossing only part of the neck.
FIG. 26. Compression wedge across entire neck.
FIG. 27. Compression wedge at the base of the neck.

effective on the head and both sides of the epiphyseal plate. In the necrotic area at the epiphyseal plate a lesser or greater part of this most important element of anchorage of the head on the neck is destroyed and the slipping of the head is made possible. The fact that besides the damaged area at the epiphyseal plate there is almost regularly a primary compression area on the head, has escaped notice and will be demonstrated.

B. In the Femoral Neck. The three described forms result if the total force of compression is spent on the head. The force, however, may continue to be effective on the femoral neck, and produce damage that manifests itself in various clinical forms.

The author found that whenever effects of compression are present in the neck, there is necessarily a primary compression area on the head also, in the segment which furnishes the fixed point during the compression. The presence of these primary foci has escaped notice and at times careful search is necessary to discover them. The author offers the demonstrable presence of these primary compression foci in support of the correctness of this concept. These primary foci are likely to be absent in younger children when the head is still covered with a thick cartilage that escapes unharmed and does not register the insult while the compression develops its damaging effect on the neck.

The femoral neck is cylindrical and the compression pattern occurring in the course of hypothetical bending is an actual wedge lying across the length axis of it. This wedge may occur at any place between the epiphyseal plate and the base of the neck. Diagrams in Figures 25 to 27 illustrate the places where the compression wedge most frequently occurs.

The clinical pictures resulting from these compression wedges in the neck are designated by different names and great confusion exists in respect to these diseases. The author has ascertained that the same findings are considered and described by one group of authors as congenital coxa vara, and as fractures of the femoral neck, by another.

R. Whitman[119] in a publication, in 1891, described these cases of juvenile fractures of the femoral neck. He clearly discerned with but little aid of radiography, that besides the frankly violent and instantaneously disabling fractures of the femoral neck, which occasionally do occur in children, and besides the epiphyseal separation known long before, there occurs not seldom in children a peculiar fracture of the femoral neck. The features of this he accurately described; arbitrarily summarized they are those of a not totally disabling pathological fracture. Irrespective of this, due to the fact that some of these findings occur quite early in childhood, a group of writers elected to describe these cases as congenital coxa vara.

The consideration of rickets and late rickets, etc., also added to the confusion.

Seen from the viewpoint of compression of cancellous bone these conditions observed in the femoral neck reveal their uniform meaning.

The necrotic bone of the compression wedge, as it undergoes mainly absorption, weakens the site and leads under weight bearing to alteration of shape and angle of the neck, or complete interruption of the continuity.

The compression wedge in Figure 25 leads to what is most often described as congenital coxa vara; those in Figures 26 and 27 are the most common Whitman juvenile neck-fracture types, the last one having been described by Taylor[106] as the "hinge fracture" of the neck of the femur of children.

COMPRESSION OF THE ACETABULUM

In the instances enumerated the compression damages the femoral head and neck. It is obvious, however, that the same amount

of pressure as on the head is also active on the acetabulum, against which the head is levered during the trauma. This often causes compression in the subchondral cancellous structure of the ace-

FIG. 28. Otto-Chroback pelvis in the Sandifort Atlas, 1793–1835.

tabulum and leads to the changes that were often observed, and have been described as osteochondritis of the acetabulum, acetabular Perthes' disease, etc. In these cases the compression may leave its mark on the head also, and the site of the latter indicates the position in which the damage originated. (Case XXII, Figs. 101 and 102.)

COMPRESSION OF THE PELVIC GIRDLE

The two hip joints, in fact the whole pelvis, may be considered a functional unit. In the course of several combinations of motions, the whole pelvic girdle may be placed under excessive stress. The existing epiphyseal plates absorb part of the pressure; but if the force is sufficient, the cancellous bone on both sides of these cartilaginous plates will be compressed in the sense of Ollier's "juxta-epiphyseal sprain." This is how the ischiopubic osteochondritis

(Van Neck),[110] and the osteochrondritis at the symphisis pubis (Burman)[24] may originate.

Compression alongside the cartilage plate of the acetabulum proper was not so far demonstrated beyond doubt, but the very controversial topic of ischium varum, and other irregularities in the relative position of the component bones of the acetabulum suggest that such compression and the following misalignment may be among the origins of the deformity named after Otto and Chroback.

This condition, too, was known considerably earlier than generally believed and is illustrated in the Sandifort[98] atlas. (Fig. 28.)

ARTHRITIS DEFORMANS OF THE HIP

The malum senile coxae, as R. W. Smith[103] conceived it, was a traumatic disease but soon it was incorporated into the group of "chronic rheumatoid arthritides," although Canton[26,27] had clearly voiced the opinion that the changes in this disease and those observed in interstitial absorption are very similar, and the former like the latter may follow a "bruise of the hip." A concise exposition of the traumatic nature of "the so-called disease rheumatoid arthritis" was forcefully given by Lane[67] in his classic essay which, although published as early as 1886, is definitely ahead of the prevailing opinion of today concerning this topic, the importance of which is amply indicated by the frequent compensation-court disputes.

Observations of early stages of arthritis deformans of the hip enabled the author to demonstrate that compression of cancellous bone is the underlying pathological process also in this condition. The typical wedge-shaped compression pattern may be found in the femoral head if observed before it collapses. (Case XXI, Figs. 99 and 100.)

This identity of nature of the process in both the senile and juvenile manifestations of interstitial absorption was also sensed by Canton.[27] Of late Putti[86] and some of his school[109] voiced the same opinion. In an earlier publication the author[16] also expressed similar belief.

COMPRESSION AT THE SACROILIAC JOINTS

During the last decade continental and American observers described changes at the sacroiliac joints, in particular at the iliac

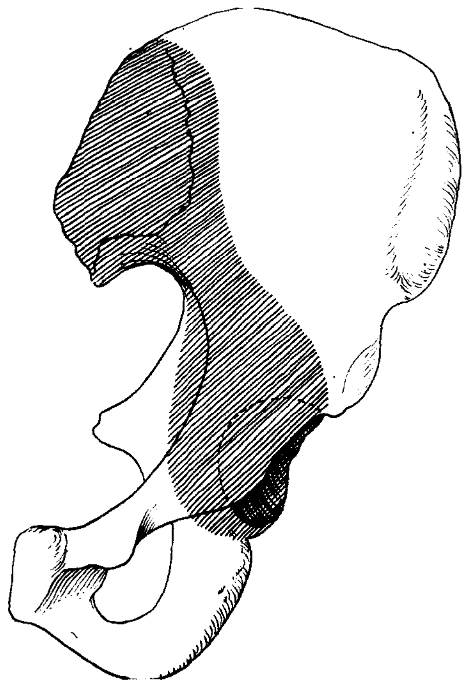


FIG. 29. The "coxo-sacral strut" in outline.

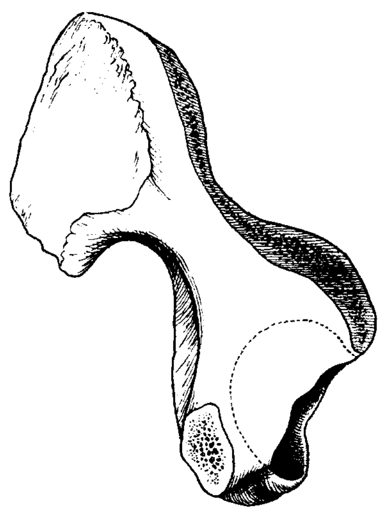


FIG. 30. The "coxo-sacral strut" shown after cutting away the nonweight transmitting portions of the pelvis.

side. These were registered somewhat arbitrarily as condensing osteitis of the ilium. (Bársony,[8] 1928, Rendich,[89] 1936.)

Irrespective of this, Rogers[95] (1935) correctly aligned well defined

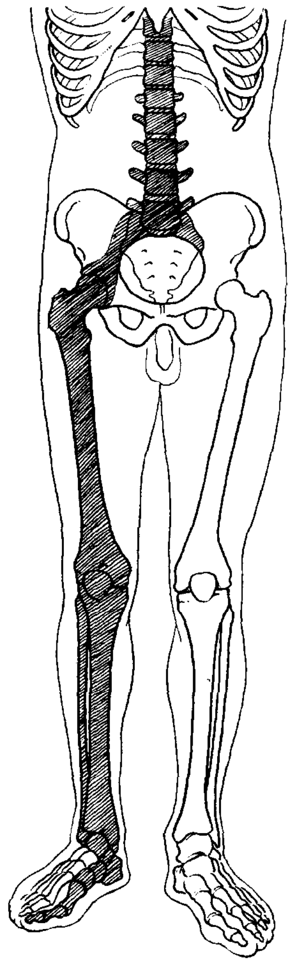


FIG. 31. The path of transference of weight from the lower extremities to the spine.

juvenile cases showing such changes with the general group of "osteochondritides as represented by Perthes' disease" and established the entity "adolescent sacroiliac syndrome."

The author shares the opinion of Rogers and, searching for the mechanism of compression, came to the following conclusion:

The weight bearing member between the femoral head and sacrum is only the solid portion of the iliac bone along the innominate line. This portion the author considers a functional unit and named it "coxosacral strut." (Figs. 29 to 31.) The stresses, strains and shocks incidental to upright weight bearing are transmitted from the lower extremities to the spine by the means of this strut. In it, proximal to the plane of transference of force, under excessive demands compression of the cancellous architecture may occur with the resulting aseptic bone necrosis. The actual presence of this necrosis the author was able to verify in drill specimens taken from these areas, as will be shown in the case reports. (Cases XXIII, Fig. 104.)

COMPRESSION OF CANCELLOUS BONE ACCOMPANYING FRACTURES

Fractures in or near segments of cancellous bone are invariably accompanied by compression of this structure, as recognized long ago. The feature which did not receive full appreciation is that in these circumstances massive necroses occur which dominate the clinical picture. Fractures of os calcis, upper end of tibia, etc., derive their protracted course from the presence of these necrotic masses which undergo absorption and substitution very slowly. Drilling is helpful in these instances, as will be reported in a separate publication.

Minute compressed foci may accompany infractions of vertebral processes and may explain a certain group of cases of "low back pain."

If it appears that the concept of the author is too widely scattered, it should be noted that it never passes beyond the boundaries of cancellous bone, not even in the instance of its most bizarre caricature, Charcot's disease. Further, it is not intended to blanket with this concept the recently described little known changes based as believed on developmental disturbances of the cartilage.

SEQUELAE OF COMPRESSION

The results of the various mechanisms of compression are identical, namely, aseptic necrosis of a portion of cancellous bone. The histological process these necrotic areas undergo, as was stated, is well known and will not be discussed at this time, with the exception of such of its aspects as, in the opinion of the author, have not been fully appraised.

Due to the accepted fact that these foci are aseptic, all-too-complete innocuity is attributed to them. Necrotic tissue within the body even if aseptic, elicits reaction from its surrounding. The reaction is not violent and consists mainly of edema. This reactive edema explains the clinical symptoms that accompany the course of all these diseases. The edema pervades the surrounding soft tissues, increases the amount of joint fluid, leads to spasm, stiffness and pain. It explains satisfactorily the paradox stiffening of joints after rest and limbering up after use. The edema spreads evenly during rest in the synovial membrane capsule, etc. With assumption of motion, functional creases have to be created in the soft parts much like in the sleeve of a new overcoat; but after the edema is squeezed out of these creases, a sense of limberness appears and increases until rest again allows the even spreading of edema and the disappearance of the creases. In the case of the hip this edema is deep and hardly palpable, but at times, as the author believes, it reveals its presence by blurring of the soft tissue markings in the *x*-ray picture. This edema persists as long as there is any trace of necrotic bone present and causes fibrosis of the muscles directly or indirectly, leading eventually to pseudoankylosis in the presence of massive foci.

This reaction, which misled some observers to suspect infection, is not instantaneous. The necrotic foci, due to the very mechanism that created them, in most instances become sealed off against the rest of the body. The compression barrier that with continued stress of use becomes more and more impenetrable, acts together with the cartilage as a closed container which prevents the necrotic material to exert its irritating effect. The reactive edema and clinical symptoms appear only when in the course of spontaneous repair vessels find their way, as demonstrated by Axhausen, around the compression barrier into the foci, and open these to the flow of blood and lymph.

This circumstance explains the period of latency that separates the injury from the clinical appearance of symptoms.

That this sealing-off actually takes place is also indicated by certain secondary changes that occur in the necrotic foci; changes that are not observed under other circumstances in necrotic bone. It seems as if in these sealed-off foci a certain interreaction of the component elements occurred, more or less physicochemically with the exclusion of biological influences. Whether calcium salts are actually in

solution there, as Leriche assumed it to be the case around fractured bone ends, and whether these salts enter into formation of insoluble calcium-soaps much like in the formation of adipocere are questions

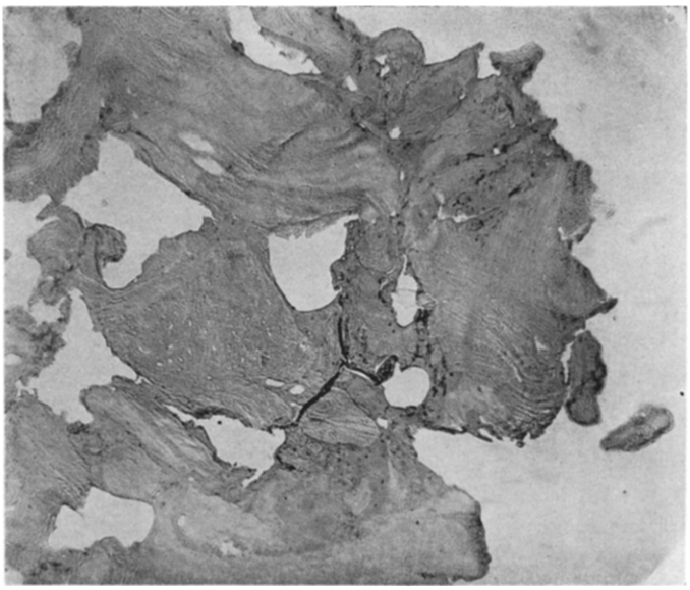


FIG. 32. Drill specimen from Case XVII. Necrotic bone with empty lacunae. Degrees of homogenization of compressed bone. Absence of living marrow elements.

of importance, particularly because, as the author had ample occasion to observe, there exists in the presence of these changes a marked degree of readiness to new bone formation. These changes lead to a marked degree of homogenization of the necrotic area, as observable on the actual specimen, and together perhaps with calcium in solution cause the blurring or loss of bone structure in the *x*-ray picture. In a former publication the author stated that these foci yield continuous ribbons of a homogeneous material that fills the grooves of the drill and is grossly and microscopically different from the occasional bone spicules obtained from living cancellous areas.

Detailed analysis of these drill specimens cannot be given in this paper. Living and dead portions of bone are clearly indicated in them, the latter not alone by the absence of any bone cells but, as was repeatedly indicated, by the complete absence of all marrow elements. (Case XVII, Fig. 32.)

FORCE

Concerning the force that produces these lesions there can be little doubt. Direct trauma acting in the direction of the neck, attacking at the greater trochanter does lead to compression in the neck and head as was clearly recognized by Bell, Canton and others. The majority of these cases, however, develop after other types of traumatism than those ordinarily designated as accidents, or after accidents that mask the participation of the hip.

As may be construed from the mechanical analysis of these lesions the force obviously acts indirectly and is multiplied by leverage in its intensity. The multiplying effect of leverage is not fully appreciated as is shown by the acts of force in reducing congenital dislocations of the hip by the closed method. The length of the limb divided by the length of the neck gives the ratio in increase of force in extreme adduction and abduction, the fulcrum assumed roughly at the intertrochanteric line.

Single insult registers itself in a clear-cut pattern of compression. Repeated trauma causes superimposed compression cones that blur and confuse the picture. The typical instance of the latter is the reduced, unreduced, and, especially, repeatedly reduced congenital dislocation.

For the very early cases the possibility of birth traumas deserve consideration. This is well recognized, because they are immediately apparent in the injuries of the upper extremities which hardly ever are subjected to such forcible handling as the lower ones. Concrete playgrounds and ball courts, and roller-skating tumbles on pavement, etc., may well account for a good number of injuries.

For the instances of arthritis deformans and the adolescent sacroiliac syndrome, of course, instead of a single insult, repeated traumas have to be assumed. Temporary or permanent loss of elasticity of gait due to fatigue or old age, with the countless small shocks it involves, may well be the causative agent. An extreme illustration of this may be seen in Charcot's disease in which the reflex muscular suspending mechanism of weight bearing is lost. The mushrooming of the cancellous substance over the condyles of the femur so often found in the aged is an illustration in lesser degree.

The intensity of force multiplied by leverage must overcome the structural strength of the cancellous segment placed under

pressure. If this strength is inferior to the average, lesser force may bring about the same compression.

The author knows of no investigations regarding physical properties of cancellous bone in the adipose-type children. Such investigations may or may not show lessened structural strength. The fact, however, that in these obese children compression damages are often observed does in itself not prove that certain endocrine disturbances do render the skeleton more susceptible to compression, because in a number of instances the same compression damages occur in known normal skeletons.

Dislocated hips in the athletic young and other perfectly normal persons, as has been known since Virchow, Zesas[124] and Maydl,[72] and which is being observed currently in a large number of cases, often show one or two typical compression foci produced by the dislocation and the reduction maneuvers, respectively.

A similar lesion was observed by the author in the head of the mandible after dislocation.

Another large group of these compression foci are observed accompanying fractures of the hip both in the intertrochanteric and, particularly, in the intracapsular types.

These compression or, as they are called, "absorption areas" are not "late damages" (Bergmann[12]) nor secondary consequences, but they are integral parts of the same injury that produced the fracture. The diagram in Figure 33 shows the author's conception of the mechanism in the case of an adduction fracture. It shows the compression wedge in the head at the site where the head is levered against the acetabulum and the compression wedge in the neck which is divided into two parts by the fracture proper.

These compression areas are at the bottom of most peculiarities this fracture represents. Concerning them, in particular those in the head, certain gross predictions may be made before they appear.

The younger the injured person and the stronger the neck of the femur, the more probable that foci will appear. Before the neck actually breaks, the head is subjected to considerable pressure, this being little or nothing in old persons whose femoral necks break easily. This circumstance accounts for the devastating absorption in the femoral head of vigorous middle aged persons, and explains that these are often observed in the present era of automobile and other violent accidents, and were seldom seen in old persons whose femoral necks break without gross violence.

These violently produced compression foci with their known date of injury support the author's conclusions about the period of latency. They will furnish us eventually with the length of period

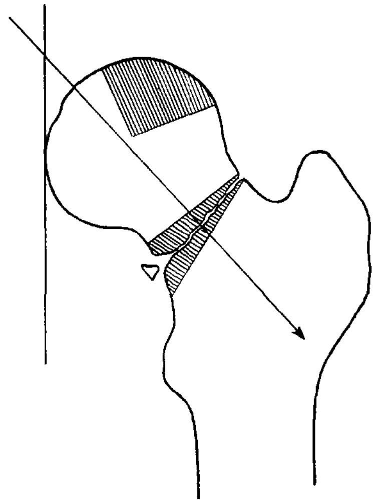


FIG. 33. Mechanism of origin of intracapsular fracture of the femoral neck in the adult. Note compression at both proximal and distal fracture surfaces and primary compression focus in the head.

that elapses between injury and clinical symptoms, that is, the onset of reaction to the necrotic tissue. At the present the author estimates this period to be four to six months.

TREATMENT

Correlating the processes of the spontaneous repair, as demonstrated by Axhausen,[6] with the concept of compression of a cancellous bone segment, led to the therapeutic principle of the author.

Axhausen[7] describes and shows the compression barrier, the "mass of bone meal" as he names it, impenetrable to nature's efforts to send capillaries across it into the foci of necrotic bone. Delitala[35] in his minute histological description of the same process tells of how capillaries "excavate a fine channel" (si scava un canale fino) across the epiphyseal plate into the head.

It appeared reasonable to expect that the penetration of the compression barrier by drill channels might facilitate the spontaneous healing process.[18] The trials were successful in a marked degree, and on their basis the author[19] came to the conclusion

published in an earlier paper that "an area of aseptic bone necrosis anywhere in the skeletal system may be advantageously treated by connecting the necrotic segment with an adjacent living segment of bone by means of drill channels."

The occurrence of compression in the femoral head and neck as previously outlined demanded that the drill channels be laid in the axis of the neck in order to penetrate any and all compressed areas. The route used by the author, the drilling from the greater trochanter, is, therefore, not merely a convenient operative approach.

Drilling, trephining or tunneling of bone in general or the femoral neck in particular, are old procedures. They arose from the convergence of Brodie's[20] classic undertaking (1832) of evacuating isolated bone abscesses by drilling into them, and the older practice of using setons to provoke productive irritation. Stanley[104] (1849), Kirkpatrick[66] (1867), J. Greig Smith[102] (1871), W. Th. Stoker[105] (1886), McNamara[74] (1887), Quénu[88] (1896), Delagénière[34] (1896), Davies-Colley[33] (1897), etc., report the extensive use of "tunneling" in the treatment of tuberculosis of the hip, of epiphyseal abscesses in the femoral neck and even of chronic (nontubercular) osteitis. Setons, on the other hand, were used to combat nonunion of fractures of long bones. The various endeavors in this field culminated in the work of Brainard[22] (1854) who not only described a procedure of drilling for nonunion of fractures of long bones but also gave a remarkably correct concept of its working principle. This work and the principle gradually became forgotten, but Loreta,[70] in 1888, performed an operation uniting the seton and drilling principle in a case of delayed union of a fracture of the base of the femoral neck by placing five metal setons and removing them after five days. Robertson Lavalle,[91,92] in 1919, described the use of bone grafts in the treatment of tuberculous osteoarthritis, and explained and demonstrated the results obtained by the penetration of new vessels from the graft into the diseased area. The drilling for nonunion was revived in 1929 by Beck[10] who, however, did not see anything "fundamentally new" in it, but a simple way of creating hematomas at the fracture site. The technical simplicity of this procedure as popularized by Boehler[14] appeared eminently adaptable for the purpose of breaking through compression barriers, and the author recommended its use in November, 1930, for instances in which such barriers presumably occurred, and performed it for the first time in January, 1931.

Among attempts of earlier date Phemister's[85] (1921) operative removal of a necrotic focus from a femoral head affected by "Perthes' disease" did not receive the merited attention. The excellent result did not lead to adoption of the clear-cut procedure, possibly because of the rôle of infection stressed by Phemister and apparently supported by operative reports of Kidner[58] (1916) and McWorther[75] (1924). Cordes,[31] in 1930, published a well conceived plan of an operation for Perthes' disease. Ferguson and Howorth,[41] in June, 1931, described a major procedure "to hasten the ossification of the epiphyseal disc," closely following the plan of Cordes missing, however, the dominant rôle of necrotic bone so clearly stressed by him.

The intimate knowledge of the repair processes following aseptic bone necrosis led to new and more correct avenues of therapeutic approach. Katzenstein[57] endeavored to influence the course of repair by transplantation of healthy bone marrow. Konjetzny[64,65] in a conscious effort to follow the course of spontaneous healing used in Koehler's disease, advised for the so-called "malacia" of the carpal semilunar osteotomies to lay open the foci to repair, a procedure which was practised by Jones[54] for the treatment of Osgood-Schlatter's disease. Zanoli[123] (1935), three years after the author, utilized drilling from the femoral head as part of a major operative intervention.

All diseases under consideration possess great tendency to spontaneous recovery. The drilling serves only to facilitate and hasten the processes of this natural healing and its ultimate aim is the removal of all necrotic tissue from the foci by these same processes. The result is commensurate with the degree in which new bone is substituted for the necrotic, but the removal of the necrotic tissue is the first postulate. As long as such portions are still present, the disease is at best latent and may flare up any time and lead eventually to arthritis deformans. The newly deposited bone, if injudiciously subjected to weight bearing and other demands, may in its turn collapse and give the disease a new start. It should be protected by all available means.

CASE REPORTS

GROUP OF OSTEOCHONDRITIS DISSECANS

Besides the osteochondritis dissecans proper, the necrotic foci developing in the femoral head after traumatic dislocations and

fractures of the neck of the femur also belong to this group by reason of their identical mechanism of occurrence (leaving out the consideration at this time of Koehler's disease, etc.).

FIG. 34. Case 1. Roentgenogram August 29, 1930; beginning separation of an osteochondritic focus.

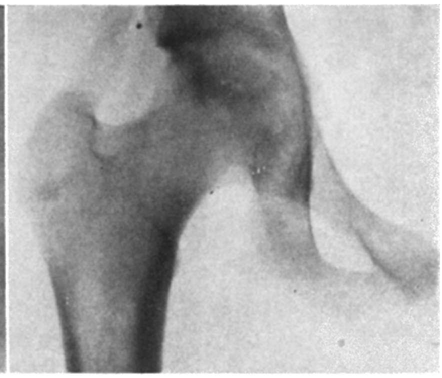


FIG. 35. Case 1. Roentgenogram January 13, 1931; nearly complete separation of sequestrum.

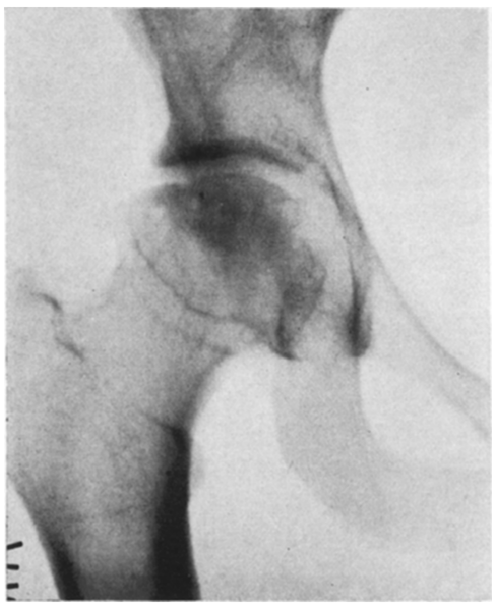


FIG. 36. Case 1. Roentgenogram five years later, March 8, 1937; fairly well preserved contour of the head; spur formation with smooth outline on upper border of the neck and head.

The material of the author falling in this group is small, but what there is indicates that the drilling should be performed as early in the course of the disease as possible.

CASE I. S. R., a twelve year old girl, had a minor bobsleigh accident, March, 1930, and a slight limp developed. *X*-ray examination August 29, 1930, showed incipient separation of a supposedly dissecting osteochondritic focus. (Fig. 34.) Subsequent *x*-ray pictures verified the diagnosis, and examination, January 13, 1931, revealed an apparently separated sequestrum in the head of the right femur. (Fig. 35.) Permission for operation was not obtained until January 31, when five channels were drilled into the head of the femur. A plaster spica was applied for a period of eight weeks. The patient was discharged May 2, 1931, with free motions and painless weight bearing in the right hip. Half-yearly checkups were made. *X*-ray examination six years later, March 8, 1937, showed a fairly well preserved head with small spur formation. (Fig. 36.) At the last clinical examination January 12, 1940, function was very satisfactory but abduction somewhat limited. Activities at work and play are unhampered. Period of observation covered nine and one-yalf years.

It seems reasonable to assume that with drilling four to five months earlier, the normal contour of the head could have been preserved and the spur formation responsible for limitation of abduction possibly avoided.

GROUP OF WALDENSTROEM-LEGG-CALVÉ-PERTHES' DISEASE

Instances of this disease present at time precarious situations in respect to spontaneous repair. When compression damages only a part of the osseous center, the repair may start from the unharmed portion. When, however, the whole center is compressed, resulting in the destruction of its vascularity and the severing of vessel connections with the surrounding cartilage, the necrotic ossification center will be cut off from all sources of capillaries; and it may be questionable whether these may reach it in time to allow both the absorption of the necrotic material and the resumption of growth of the head.

This circumstance explains the amazing chronicity of cases of this disease at a period of life when repair is the swiftest and when foci of identical nature and size in more accessible places of the skeleton are eliminated in a few weeks; and this accounts mainly for the fact that necrotic portions may remain in the head for eight or ten years unabsorbed and unsubstituted as was found by Konjetzny.[64,66]

The drilling into the necrotic center of the head is of striking benefit. Its effects develop in two phases that are fairly well sepa-

rated. First, a swift absorption of all necrotic material occurs; subsequent to this the normal growth of the head is resumed. The first phase is indicated by the appearance of marked contrast within the ossification center, due to absorption of necrotic portions. The absorption completed, the second phase or normal growth of the head starts on its way. The ultimate outcome depends on, and may be predicted by the amount of cartilage present at the time of drilling. The greater this *cartilage reserve* the more perfect the end result. This demands that the drilling be done as early as possible. If the cartilage reserve is small, the drilling is of even greater urgency, because a thin cartilage covering is not sufficient to cushion against further deformation and itself may wear through and so lead to the destruction of the head.

The appearance of the head during the first phase may be quite alarming at times. Fragmentation may become more pronounced than it was before the drilling. Observations during the last seven years convinced the author that this increased fragmentation is the result of absorption of necrotic parts and indicates no danger. As long as there is a thick cartilage layer over the bony center, however badly fragmented, the outcome will be good. Arthrography as described by Sievers[101] and Severin[100] will remove the guess work as to the amount of cartilage reserve, but this may be calculated correctly enough on the usual *x*-ray picture. In the second phase a wedge-shaped defect may appear on the fairly well developed head. This, as stated, marks in the opinion of the author the area in the cartilage where compression damaged the vessels and ossification is consequently retarded. These areas persist for a long time, but eventually become filled up and do not necessitate a second drilling. The flat appearance of the head, besides its actually lesser height, is in a measure due to the slipping of the head in the same manner as observed in the cases of slipped epiphyses. This circumstance, although noted by Waldenstroem[113] in his first publication (1909), is not generally recognized; it is clearly demonstrated in Figures 50, 53 and 59.

As a basic plan the author recommends accurate drilling as early as possible, plaster of paris spica immobilization for six to eight weeks, protective brace until *x*-ray examination proves that all necrotic portions were absorbed. Beyond this period, in the opinion of the author, protection against weight bearing is no more necessary than it is for the femoral heads of healthy children during

normal growth. For the time being, however, until more material can prove the safety of this view the author uses and recommends the use of a brace as long as the children can be induced to wear it.

FIG. 37. Case 11. Roentgenogram April 26, 1933; irregular head with flattened contour and abnormal structure; broadened neck.

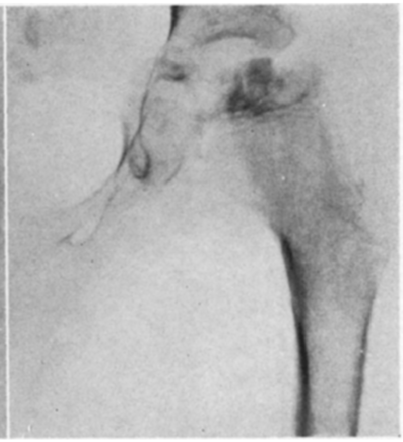


FIG. 38. Case 11. Roentgenogram five weeks after drilling, June 21, 1933; marked absorption of portions in the head and contrasting structure.

CASE 11. P. W., a seven year old boy, gave a history of pain, limping and stiffness in the right hip for half a year. On the basis of clinical and repeated x-ray examinations the diagnosis of Waldenstroem-Legg-Calvé-Perthes' disease was made. (Fig. 37.) A plaster of paris spica for three months brought no relief. The drilling operation was performed on May 11, 1933, and a short knee-length spica applied. The patient was discharged five days postoperatively. Six weeks after the operation the spica was removed. *X*-ray examination the following day showed that marked contrast developed in the ossification center, and portions of it became absorbed. (Fig. 38.) A brace was recommended, but the patient was not heard of until three and one-half years later, in February, 1937, when the parents stated that, as the child walked about without pain, no brace was used. The child at that time had no symptoms whatever, attended school, and participated in all exercises during the three and one-half years that elapsed. *X*-ray examination February 12, 1937, showed the femoral head fairly well developed, with a crater-shaped defect of ossification in its middle portion. (Fig. 39.) Half-yearly checkups were made. *X*-ray examination February 8, 1940, (Figs. 40 and 41) shows well shaped and developed head both on the anteroposterior and lateral views. A faint trace of the one-time crater is indicated. Range of motion was complete. The period of observation lasted seven years.

Case iii. J. B., a five and one-half year old boy, had pain in the right hip, a limp and increasing inability to walk for a period of eight months. Hospital observation and prolonged bed-rest at home were carried out but

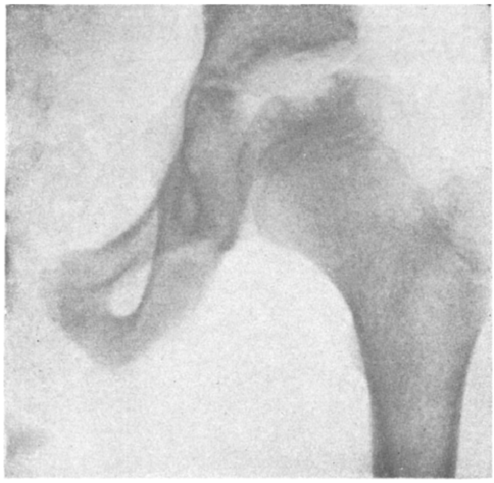


Fig. 39. Case ii. Roentgenogram three and three-quarter years after drilling; development of head well under way; crater of retarded ossification at site of vessel damage in the cartilage.

Fig. 40. Case ii. Roentgenogram six and three-quarter years after drilling, February 8, 1940; well developed head of good structure.

Fig. 41. Case ii. Lateral view same day as Figure 40; well developed head; trace of crater faintly perceptible.

there was no improvement. *X*-ray examination on May 19, 1934, showed an area of bone destruction in the upper-outer segment of the head and neck. (Fig. 42.) The child was kept in bed at home without marked improve-

ment, and permission for operation was not obtained until September 8, 1934, when three channels were drilled into the head in the direction of the focus. No spica was applied. Six weeks rest in bed followed. *X*-ray exami-

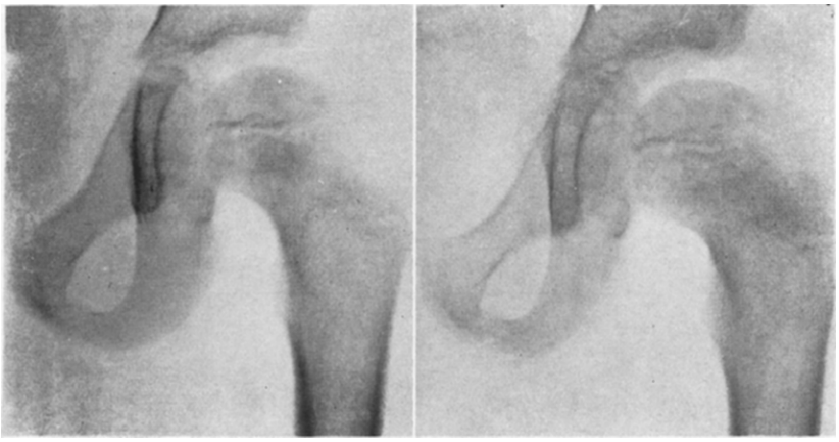


FIG. 42. Case III. Roentgenogram May 19, 1934; area of bone destruction indicated in upper-outer segment of head and neck.

FIG. 43. Case III. Roentgenogram six weeks after drilling, October 25, 1934; marked absorption in the focus indicated in Figure 42, with contrast in bone structure.

nation on October 28, 1934, showed a well developed contrast at the site of the focus with marked absorption of the involved area. (Fig. 43.) A brace was worn for six months. The child reported to follow-up only two and one-half years later, when the parents stated that after the brace was discarded the child continued with his activities without symptoms. The child has been seen yearly since then. *X*-ray examination February 10, 1939, showed well shaped and developed head on both the anteroposterior and lateral views. Epiphyseal line showed irregularity at the site of the original focus but no other abnormality. (Figs. 44 and 45.) Range of motion is complete. The period of observation lasted five years.

CASE IV. R. D., five year old boy, had pain in the left hip, a limp and inability to stand and walk, for a few weeks. *X*-ray examination of the left hip on November 24, 1934, showed a typical picture of Waldenstroem-Legg-Calvé-Perthes' disease. (Fig. 46.) The drilling operation was performed on November 28, 1934, followed by bed-rest in the hospital for eight weeks. *X*-ray examination on February 4, 1935, showed what was then thought very alarming fragmentation of the ossification center of the head (Fig. 47), and for this reason a plaster of paris spica was applied for three months. After removal of the spica a brace was applied. The child was seen every two months and while the *X*-ray pictures still looked very discouraging, the clinical symptoms gradually disappeared and the child walked

FIG. 44. Case III. Roentgenogram four and one-half years after drilling, February 10, 1939; no trace of abnormal structure, well developing head; neck somewhat broadened.

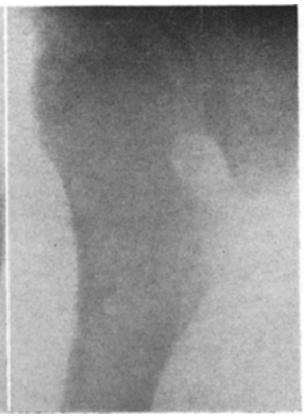


FIG. 45. Case III. Lateral view, same day as Figure 44; irregularity of epiphyseal line at site of focus; no other abnormality.

FIG. 46. Case IV. Roentgenogram November 24, 1934; typical findings of early Legg-Perthes' disease.

FIG. 47. Case IV. Roentgenogram seven weeks after drilling, February 4, 1935; alarming fragmentation of the head; two drill channels visible.

about without concern and the brace performed obviously no function whatever except perhaps restraining his activities. *X*-ray examination on October 4, 1935, still showed marked fragmentation, but something of a

FIG. 48. Case IV. Roentgenogram thirteen months after drilling, February 6, 1936; growth of head well under way.

reshaping of the ossification center could be perceived. At this time the child was entirely without clinical symptoms and used the brace only while in school. The brace was discarded when *x*-ray examination, February 6, 1936, showed that the regrowth of the head was definitely on its way. (Fig. 48.) Follow-up examinations were made at half-year intervals. *X*-ray examination showed a steady growth of the head. The last *x*-ray examination on February 13, 1940, showed the femoral head well shaped and developed and the neck of good length without broadening. (Fig. 49.) Lateral view shows slight off-center position of the head. (Fig. 50.) Range of motion is complete. The period of observation covered five and one-quarter years.

CASE V. A. L., seven year old boy, gave a history of pain and limp in the left hip. *X*-ray examination on November 9, 1934, showed irregular ossification center and epiphyseal line of the left hip. (Fig. 51.) The diagnosis of Waldenstroem-Legg-Calvé-Perthes' disease was made and the drilling operation performed on November 16, 1934. A plaster of paris spica was applied for eight weeks but no brace. Home conditions necessitated hospitalization for two additional months, after which the child was transferred to an orphan home where he was under constant supervision. Half-yearly checkups were made. *X*-ray and clinical examinations showed steady growth of the head and absence of symptoms. The last *x*-ray examination on February 10, 1940, showed a fairly well shaped and developed

head. (Fig. 52.) Range of motions is complete and the child is pursuing all activities at school. The reduced height of the head as seen on the antero-posterior view is to a great extent only apparent and is due to the slipped

FIG. 49. Case IV. Roentgenogram five and one-quarter years after drilling, February 13, 1940; well developed head and neck, normal bone structure.

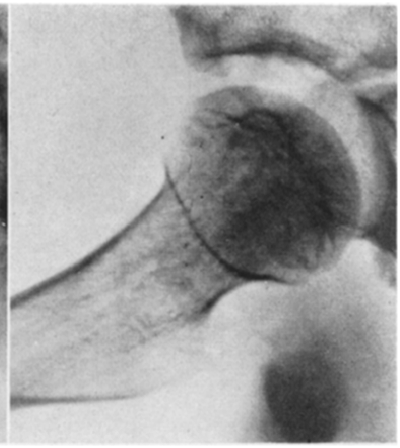


FIG. 50. Case IV. Lateral view, same day as Figure 49; head is slightly off center.

FIG. 51. Case V. Roentgenogram November 9, 1934; early Legg-Perthes' disease.

FIG. 52. Case V. Roentgenogram five and one-quarter years after drilling; well developing head somewhat off center.

position of the head as it is plainly noticeable on the lateral view. (Fig. 53.) The period of observation covered five and one-quarter years.

CASE VI. L. L., a five and one-half year old boy, had marked pain in the left hip, a limp, and inability to walk, except short distances for a

period of four months. X-ray examination February 6, 1935, corroborates the clinical diagnosis of Waldenstroem-Legg-Calvé-Perthes' disease. (Fig. 54.) The drilling operation was done on February 19, 1935. A spica was

FIG. 53. Case v. Lateral view, same day as Figure 52; head well shaped and developed but in a marked off center position.

applied for eight weeks and a brace for fourteen months. X-ray examination April 15, 1935, showed marked contrast developing in the ossification center of the head assuming the appearance of osteochondritis dissecans. (Fig. 55.) X-ray examination on July 10, 1935, showed the full development of this subcartilaginous osteochondritis dissecans. Sequestrum and its bed from which it separated were plainly visible. (Fig. 56.) Clinical symptoms steadily decreased and the child became more and more active. February 26, 1936, a year after drilling, x-ray examination showed that while a small portion of the sequestrum still persisted, the growth of the head was definitely under way. The child could be restrained only with great difficulty, but owing to the fact that the osseous center of the head was apparently off center, an endeavor was made to continue with the brace, which was discarded only sixteen months after the operation. Half-yearly checkups were made. X-ray examination September 11, 1936, showed progressing growth of the head; a small portion of the sequestrum was still visible, the outlines of compression crater indicated on the head. (Fig. 57.) The child was last seen February 10, 1940, when the x-ray examination revealed a well shaped and developed neck and head (Fig. 58), the latter somewhat displaced posteriorly as indicated on the lateral view. (Fig. 59.) The period of observation covered five years.

FIG. 54. Case VI. Roentgenogram February 6, 1935; typical picture of early Legg-Perthes' disease.

FIG. 55. Case VI. Roentgenogram nine weeks after drilling, April 15, 1935; absorption in the head bringing about picture of osteochondritis dissecans.

FIG. 56. Case V. Roentgenogram July 10, 1935; picture of osteochondritis dissecans fully developed.

Fig. 57. Case vi. Roentgenogram a year and one-half after drilling, September 11, 1936; growth of head resumed, vestige of sequestrum visible; compression crater in head outlined.

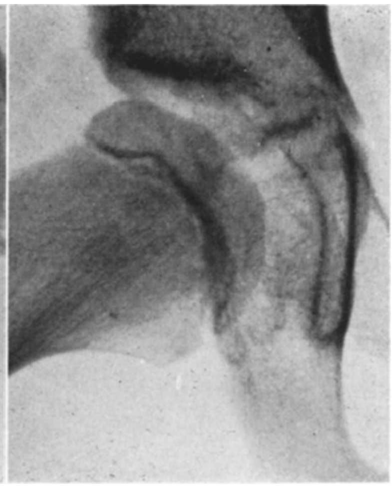


Fig. 58. Case vi. Roentgenogram five years after drilling, February 10, 1940; well shaped and developed head and neck; normal bone structure.

Fig. 59. Case vi. Lateral view, same day as Figure 58; head slightly off center.

CASE VII. J. E., a seven year old boy, had marked pain and spasm in the right hip, inability to stand on right leg and to walk any but short distances. The duration of symptoms was ten months. On clinical and *x*-ray

FIG. 60. Case VII. Roentgenogram seven months after drilling, December 24, 1935; head still fragmented, compression crater clearly outlined.

FIG. 61. Case VII. Roentgenogram four and three-quarter years after drilling, February 8, 1940; well developed head and neck; normal bone structure.

FIG. 62. Case VII. Lateral view, same day as Figure 61.

evidence the diagnosis of Waldenstroem-Legg-Calvé-Perthes' disease was made. (*X*-ray pictures of that period cannot be located.) The drilling operation was performed on May 23, 1935. A plaster of paris spica was applied followed by bed-rest for seven months. *X*-ray examination on

December 24, 1935, showed the ossification center of the head still fragmented, the outlines of a crater indicated on its middle. (Fig. 60.) Subsequent *x*-ray examinations showed filling of this defect and fusion of the fragmented parts within one year and half postoperatively. For reasons of precaution rest in bed alternating with spica was continued for another ten months after which the child resumed normal activities. For one and three-quarter years the child could not be located, but when found was well and active, *x*-ray examination showing well developing head. He was last seen February 8, 1940, when the *x*-ray examination at that time showed a well shaped and developed head and neck on both anteroposterior and later views. (Figs. 61 and 62.) Range of motion was complete. The period of observation lasted five years.

GROUP OF SLIPPING EPIPHYSES

As was stated, the underlying cause of the slipping of the head is the necrosis at the epiphyseal plate, in particular, distal to it. Continued weight bearing often frustrates spontaneous repair by crushing the newly formed bone. When pain and spasm intermittently immobilize the patient, absorption of the necrotic parts and the deposition of new bone may be so well balanced that the head may wander great distances on the neck without actually separating from it.

Many features of this disease may be explained by simple mechanical interpretation. The head wanders downward and onto the posterior surface of the neck because the place of easiest escape is the intertrochanteric fossa. One glance at the upper end of the femur from above fully reveals this situation. Femoral heads after complete and incomplete neck fractures invariably follow the same direction.

Owing to this direction of slipping, the synovial membrane at the lower surface of the neck lifts into a fold. This fold is part of the normal synovial membrane and not the result of inflammatory redundance. Its inflamed appearance is due to mechanical irritation in the crowded joint, and not, as at times it was believed, to "primarily infectious" reasons.

In the same way as the synovial membrane, the periosteum of the neck may also lift into a fold at the lower contour of the neck and produce there new bone, as has been observed since the days of Bell, Cooper and Canton.

As these soft part coverings of the neck fold below and behind, they become tautly stretched in front and above. At times they

thicken and produce cartilage patches, at others they wear away and the bone and epiphyseal cartilage may become exposed. Particles of the latter may be rubbed off into the synovial membrane or into the free joint, thus bringing within the possible consequences the condition called chondromatosis.

The sudden and complete separations need no further explanation with the exception that it is not generally realized that upright weight bearing is one of the mechanical factors that tend to keep the head on the neck. Sudden complete slips while asleep are explained by the absence of this stabilizing element and by the direction of force across the neck when shifting the body in a horizontal position.

Drilling in the instances of this disease brings uniformly good results. The necrotic areas are rapidly absorbed, the epiphyseal plate disappears in most instances, and head and neck fuse firmly. Drilling freezes, as it were, the existing situation. The author recommends six to eight weeks' plaster of paris spica immobilization after operation, with subsequent brace protection against weight bearing. The larger the area of necrosis at the time of the drilling, the longer should be the period of brace protection. In cases of complete separation protection against weight bearing should be practiced for prolonged periods, possibly two years or more, and the condition carefully checked by *x*-ray examination at quarterly intervals. Any ill conceived hurry to produce spectacular results may end in the disaster of deforming arthritis. If diagnosis will be made early, as emphatically demanded by Mayer,[73] and drilling performed as soon as possible, the results will be swift, perfect and permanent.

The outcome of neglected cases is at times appalling. Arthritis deformans with pseudoankylosis lead to a great degree of permanent disability. At times a curious and little understood condition results as illustrated in Case XVI. A prominent feature of this condition is that the femoral head is markedly enlarged.

Andral[3] reported the condition as an accidental autopsy finding in 1834. It was again briefly outlined by John Sandifort,[127] in 1840, as "augmented volume" of the femoral head. In 1868, Volkmann[112] described it as "true hypertrophy" of the femoral head and attributed it to increased blood supply. Guibal[50] called it "deforming and hypertrophying osteochondritis" in 1922. Recently this condition was described and renamed "coxa magna" by Ferguson and Howorth[126] (1935).

CASE VIII. T. H., a ten year old boy, was first seen July 10, 1931. He had pain and stiffness in the left hip. *X*-ray examination at that time showed slight disturbance of structural detail distal to the epiphyseal plate interpreted as necrosis. (Fig. 63.) No permission for drilling was obtained until *x*-ray examination, November 10, showed marked progress of the disease. (Fig. 64.) The operation was performed November 30, 1931. Five channels were drilled into the neck and head. A spica was applied for eight weeks and a brace for ten months. The patient and parent were very co-operative and the child was restrained from strenuous exercises for a second year during which, however, he attended school and was entirely symptomless. Half-yearly checkups were made. *X*-ray examination February 17, 1937, showed a well shaped and fully developed head and normal bone structure. Shortening of the neck was proportionately the same as at the time of drilling. (Fig. 65.) The patient was last seen June, 1939, when the range of motion was complete. The period of observation lasted eight years.

FIG. 63. Case VIII. Roentgenogram July 10, 1931; early disturbance of bone structure in neck at epiphyseal plate.

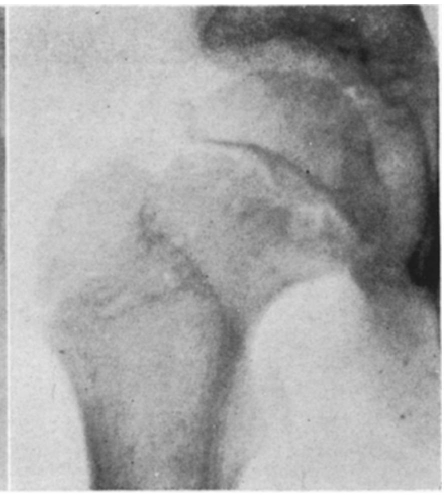


FIG. 64. Case VIII. Roentgenogram November 10, 1931 marked progress of the disease. Vestige of primary compression in head.

CASE IX. M. McE., a ten year old girl, gave a history of pain and limp of one week. *X*-ray examination on October 19, 1933, showed marked bone destruction in the neck without markedly changed position of the head. (Fig. 66.) After two weeks of traction treatment in bed, the drilling operation was performed October 25, 1933. A spica, first full, then knee length for fifteen weeks was applied. This prolonged treatment in a spica resulted in a moderate stiffening of the knee on account of which rest and careful exercise in bed were carried out till the end of the sixth month after

the operation, when the patient was permitted to walk on crutches. These were gradually discarded. Half-yearly checkups were made during which her condition steadily improved functionally and as to *x*-ray appearance.

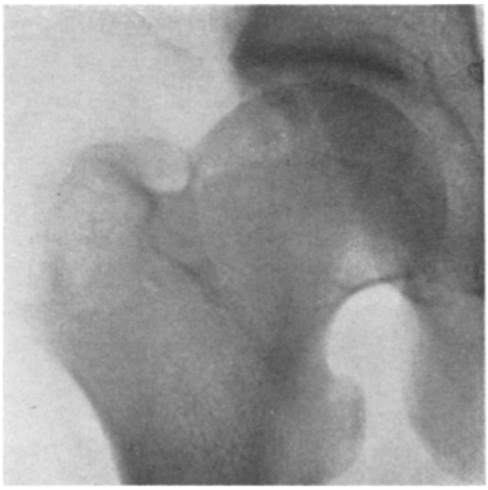


FIG. 65. Case VIII. Roentgenogram five years after drilling, February 17, 1937; well shaped and developed head; normal bone structure; fusion of epiphyseal plate.

FIG. 66. Case IX. Roentgenogram October 19, 1933; marked bone destruction in the neck.

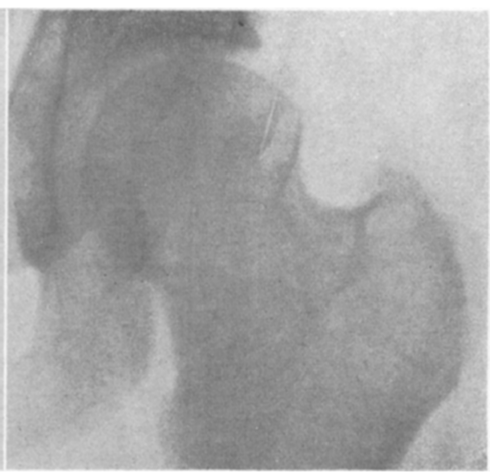


FIG. 67. Case IX. Roentgenogram three and one-quarter years after drilling, March 6, 1937; complete fusion of head and neck; normal bone structure; well shaped head.

The last *x*-ray picture, March 6, 1937, showed a perfectly shaped and developed head with normal bone structure. (Fig. 67.) The range of motion was complete. The period of observation covered six and one-quarter years.

Case x. M. P., a twelve year old girl, was first seen August 1, 1933. She gave a history of pain in the left hip for a period of a month and walked with a marked limp. An *x*-ray picture taken on that day showed

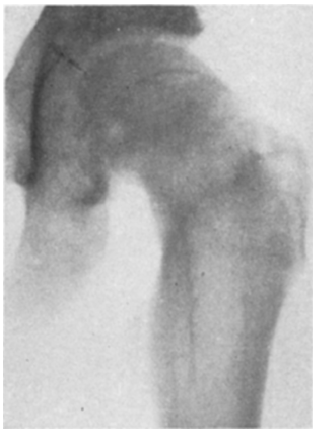


Fig. 68. Case x. Roentgenogram August 1, 1933; marked destruction in the neck; definite slipping of the head.

Fig. 69. Case x. Roentgenogram three and one-quarter years after drilling, March 6, 1937; complete fusion, well shaped head; normal bone structure.

marked destruction in the neck distal to the epiphyseal plate and decided slipping of the head. (Fig. 68.) A plaster of paris spica was applied for two months. No marked improvement took place. The drilling operation was performed on October 11, 1933 and a spica applied for five weeks. Careful exercises and walking on crutches were then permitted. After six months crutches were discarded. The child walked without limp or pain. Half-yearly checkups were made. The last *x*-ray examination, March 6, 1937, showed a perfectly shaped and developed head with normal bone structure. The position of the head was the same as at the time of the drilling and very satisfactory; length of neck was proportionately the same as at the time of drilling. (Fig. 69.) Range of motion was complete. The period of observation lasted five and one-half years.

Case xi. F. S., an eleven year old girl was first seen August 22, 1934. She gave a history of pain, spasm in the left hip and inability to walk any but short distances. *X*-ray examination showed bone destruction at the epiphyseal plate and beginning displacement of the head. Drilling operation was performed on August 23, 1934, but no spica was applied. Due to circumstances over which the author had no control, the patient left the hospital nine days after operation and was allowed to walk about. On February 6, 1935, the patient again returned with a marked limp and pain. *X*-ray examination at that time showed marked progress of the disease.

(Fig. 70.) A second drilling was recommended. February 21, 1935, five channels were drilled in the neck and head. A spica was applied for eight weeks and a brace for nine months. The condition rapidly improved.

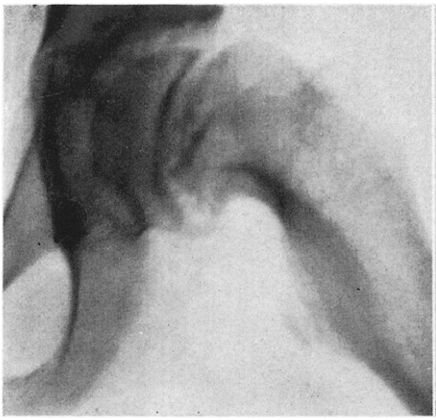


FIG. 70. Case XI. Roentgenogram February 6, 1935; marked slipping of the head and bone destruction in the neck.

FIG. 71. Case XI. Roentgenogram five years after second drilling, December 30, 1939; complete fusion, well shaped head; normal bone structure.

FIG. 72. Case XI. Lateral view, same day as Figure 71; shape of head well preserved.

Subsequent *x*-ray examinations showed good repair. One year after the second drilling the child was fully active. Half-yearly checkups were made. The last *x*-ray examination on December 30, 1939, five years after drilling, showed the head in the same relative position as at the time of drilling, well shaped and of normal structure. (Figs. 71 and 72.) Range of motion was very satisfactory. There was a trace of limitation of abduction and internal rotation. The period of observation covered five and one-half years.

CASE XII. J. K., a seventeen year old boy, suffered with disability of the left hip for one and one-half years. *X*-ray examination May 23, 1932, showed marked coxa vara and bone destruction distal to the epiphyseal

FIG. 73. Case XII. Roentgenogram May 23, 1932; marked coxa vara, bone destruction in the neck, off center position of the head; primary focus indicated in head.

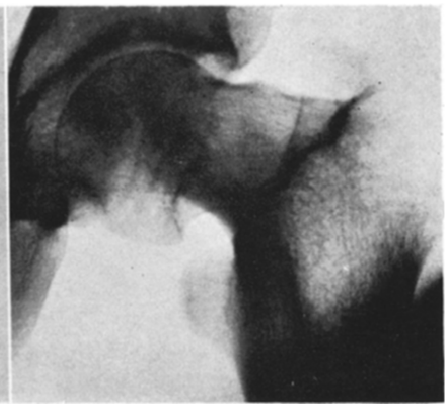


FIG. 74. Case XII. Roentgenogram one-half year after drilling, November 10(?), 1932; firm fusion; primary focus still outlined in head.

FIG. 75. Case XIII. Roentgenogram February 7, 1932; complete epiphyseal separation of right hip.

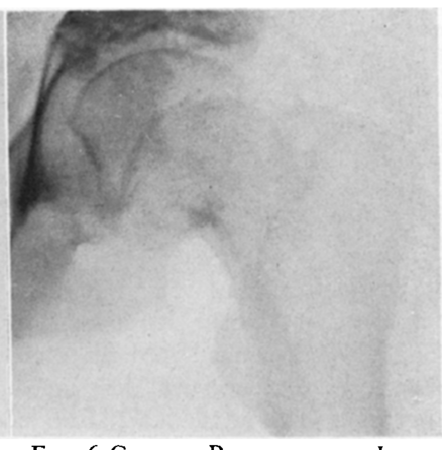


FIG. 76. Case XIII. Roentgenogram three months after drilling, May 7, 1932; maintained continuity between head and neck; definite primary compression focus in head.

plate. (Fig. 73.) Drilling operation was performed June 6, 1932, and a spica applied for eight weeks. Under the care of a private physician measures of protection against weight bearing were carried out. *X*-ray examination on November 10(?), 1932, showed firm fusion of head and neck. (Fig. 74.)

Osteotomy for the correction of the coxa vara was recommended but the patient did not return. The period of observation was six months.

CASE XIII. B. S., a ten year old girl, stumbled and fell on January 12,

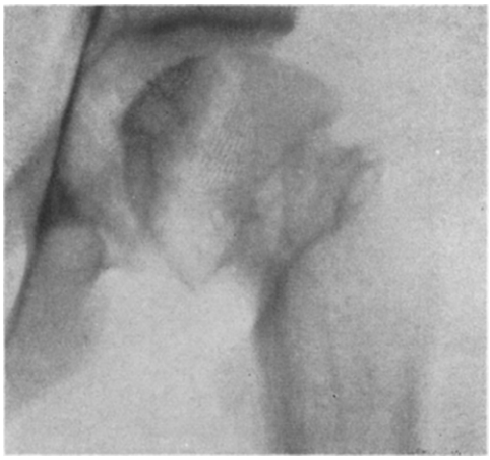


FIG. 77. Case XIII. Roentgenogram five years after drilling, February 3, 1937; firm union, fairly well shaped head; primary focus in head still persisting.

1932. Her right lower limb was instantly disabled and she was kept in bed at home. On February 7, 1932, *x*-ray examination showed complete epiphyseal separation in the right hip. (Fig. 75.) On February 10, 1932, the separation was reduced by the author's method of lateral traction, and the drilling operation performed. A spica was applied for the period of eight weeks. Rest in bed and careful exercise were prescribed. On May 7, 1932, *x*-ray examination showed maintained continuity of neck and head and the appearance in the latter of a characteristic primary focus of compression which was not noticeable in the previous pictures. (Fig. 76.) A brace was applied for six months and the child was transferred to a convalescent home. Yearly checkups were made. *X*-ray examination on February 3, 1937, five years later, showed firm union and a fairly well shaped head. The vestiges of primary compression focus were still clearly discernible. (Fig. 77.) Motions were slightly limited in all directions, but the range was satisfactory. She was last seen on February, 1940, when her condition was the same. The period of observation lasted eight years.

CASE XIV. B. S., the same patient as Case XIII. *X*-ray examination on February 7, 1932, was taken of the left hip incidentally with the injured right hip, but revealed no pathological findings. (Fig. 78.) On June 5, 1932, the left hip was again included in the *x*-ray picture taken as a follow-up of the right hip. This revealed a similar compression focus on the left femoral

head (Fig. 79) as was noted on the right. For this reason the author recommended bed-rest immobilization, possibly drilling of the left hip. This was not carried out and the child was permitted to walk about with a brace on

FIG. 78. Case XIV. Roentgenogram February 7, 1932. Normal head and neck of left femur (same patient as Case XIII).

FIG. 79. Case XIV. Roentgenogram July 5, 1932; definite primary compression focus in head.

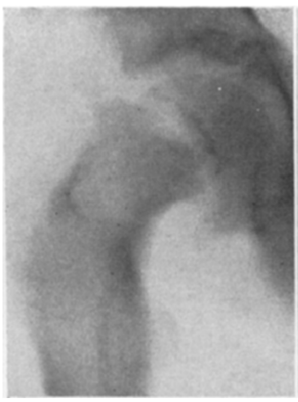


FIG. 80. Case XIV. Roentgenogram October 14, 1932; complete epiphyseal separation, left hip.

FIG. 81. Case XIV. Roentgenogram five years after drilling, February 15, 1937; firm union, fairly well shaped head; primary compression focus still visible.

the right leg with activities moderately restricted. On October 14, 1932, the patient returned with a completely slipped epiphysis in the left hip. (Fig. 80.) Reduction and drilling were performed on October 25, 1932, and a spica applied for eight weeks. The child returned to the convalescent home. Yearly checkups were made. *X*-ray examination on February 15, 1937, four and one-quarter years after drilling, showed firm union and a fairly well shaped head. Vestiges of primary compression focus were still discernible.

(Fig. 81.) Motions were somewhat limited in the right hip, but range was satisfactory. She was last seen February, 1940, and her condition was the same. The period of observation lasted seven and one-quarter years.

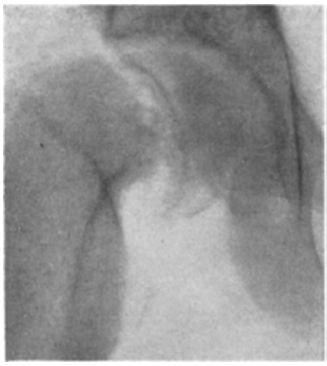


FIG. 82. Case xv. Roentgenogram April 14, 1934; complete epiphyseal separation, left hip.

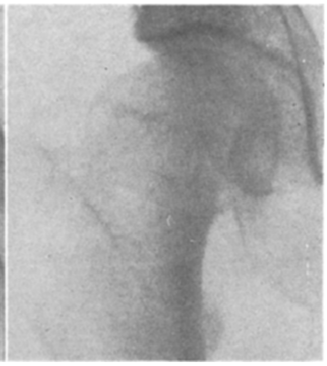


FIG. 83. Case xv. Roentgenogram two and one-half years after drilling, November 6, 1936; firm union, fairly well shaped head in fair position; normal bone structure.

FIG. 84. Case xvi. Roentgenogram August 1, 1933; old slipped epiphysis with some amount of union; head markedly enlarged and of abnormal structure.

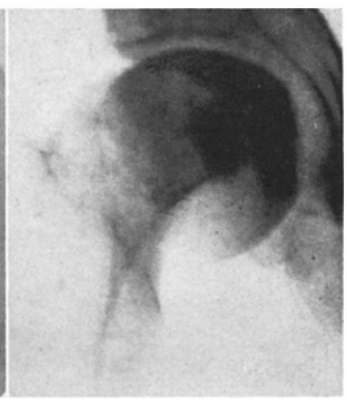


FIG. 85. Case xvi. Roentgenogram eight years previous to Figure 84, January 26, 1926. Original condition: slipping epiphysis.

CASE XV. J. V., a fourteen year old, obese boy fell at home and his left lower extremity was instantly disabled. He remained in bed for two weeks. *X*-ray examination, April 14, 1934, showed a completely slipped epiphysis in the left hip. (Fig. 82.) On the same day reduction and drilling were performed. A spica was applied for eight weeks and brace protection for six

months. His left knee developed a stubborn stiffness that yielded completely only after eighteen months. Constant supervision was carried out. *X*-ray examination, November 6, 1936, showed firm union and normal bone structure. The position of the head was satisfactory. (Fig. 83.) Range of motion was slightly limited in every direction. He was last seen March, 1937. The patient moved but reports by letter indicate good function. The period of observation covered two and one-half years.

Case xvi. A. H., a twenty-five year old male, gave the history of long disability of the right hip. According to his statement he suffered from "slipping epiphysis" and was treated at several hospitals. Spica and bed-rest with traction were used. *X*-ray examination on August 1, 1933, revealed an old epiphyseal slip with union, the extent of which cannot be well determined. The head appeared enlarged and of markedly abnormal structure. (Fig. 84.) Weight bearing in right hip was fair but painful. The drilling operation was performed on April 30, 1934, after which the patient left city and no follow-up was possible. On inquiry at the hospitals where patient was previously treated it was found that an *x*-ray examination was made January 26, 1926, which proved that the original condition was slipping epiphysis of the right hip. (Fig. 85.) During the seven and one-half years that elapsed between the two *x*-ray examinations the condition progressed from slipping epiphysis to what was lately termed coxa magna, demonstrating the relationship between the two.

GROUP OF COMPRESSION FOCI IN THE FEMORAL NECK

Case xvii. E. G., a five year old boy, gave a history of trivial tumbles while playing during summer, 1931. No definite accident occurred. There was some pain in the right hip, a very marked limp, reluctance to walk and total inability to stand on the right leg. *X*-ray examination, December 10, 1931, revealed a large triangular subcapital segment of the neck separated without displacement. (Fig. 86.) This segment had the appearance of necrosis. The course of illness was afebrile and the patient was kept in bed. On December 22, 1931, two channels were drilled, one into the axis of the neck and the other into the affected segment. The limb was immobilized. The bone specimen removed in the grooves of drill showed total necrosis. (Fig. 32.) *X*-ray examination on January 28, 1932, five weeks after operation, showed re-union of the detached portion with the neck, the previously necrotic segment evidently being re-vitalized. The two drill channels were visible. (Fig. 87.) Clinical recovery was rapid and complete. *X*-ray examination one year after operation showed no disturbance and good growth. The patient was seen regularly for two more years. He is well and active. The period of observation lasted three years.

Case xviii. H. C., an eight year old boy, gave a history of long disability. The mother stated, "both hips were broken when baby was

dropped." *X*-ray examination on January 5, 1932, of the left hip showed marked coxa vara with displacement of the head below the greater trochanter and the neck at a sharp angle with the shaft. Across the neck a

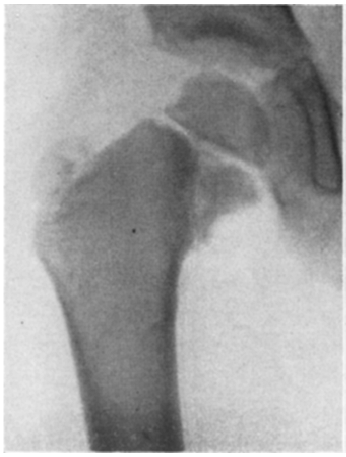


FIG. 86. Case XVII. Roentgenogram December 10, 1931; necrotic subcapital segment separated but not displaced.

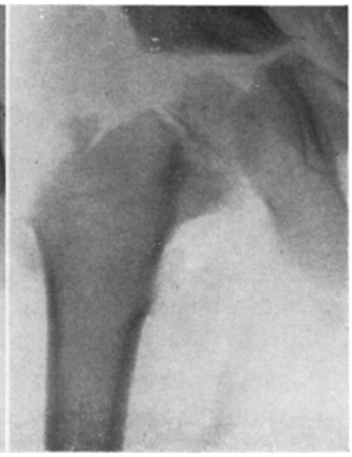


FIG. 87. Case XVII. Roentgenogram five weeks after drilling, January 28, 1932; necrotic segment fused to neck; two drill channels visible.

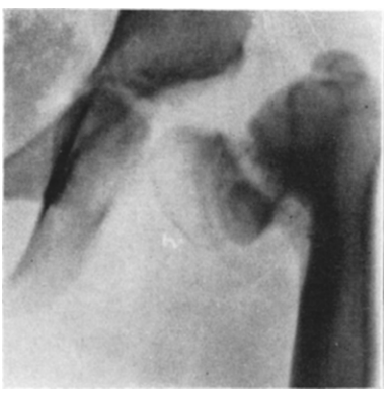


FIG. 88. Case XVIII. Roentgenogram January 5, 1932; marked coxa vara; broad defect across neck.

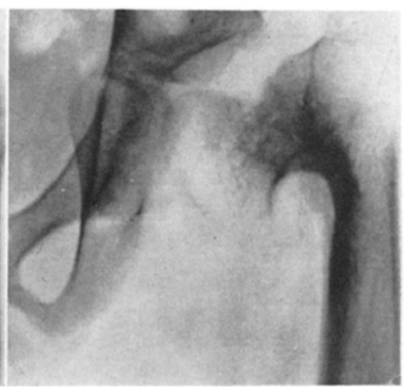


FIG. 89. Case. XVIII. Roentgenogram June 3, 1932; bony union at the site of the previous bone defect.

broad band of absorption interrupts the continuity outlining a wedge-shaped segment distal to the epiphyseal plate. (Fig. 88.) The same condition was observed in the right hip, on which osteotomy was performed on January 14, 1932. On the occasion of change of spica, January 28, 1932, three channels were drilled through a small incision over the greater tro-

chanter into the left femoral neck. Owing to the presence of spica no x-ray picture was taken of the left hip until June 3, 1932. At that time complete bony consolidation was present at the site of the area of absorption that

FIG. 90. Case XVIII. Roentgenogram five years after drilling and osteotomy, March 8, 1937; well developed head and neck.

existed prior to the drilling. (Fig. 89.) Subtrochanteric osteotomy was performed for the correction of coxa vara on September 22, 1932. Half-yearly checkups were made. After June, 1934, the child was irregular in reporting and was not located until March 8, 1937. An x-ray examination at that time showed a fairly well developed femoral neck in good position, normal head, and satisfactory bone structure. (Fig. 90.) The child was not very co-operative and could not be restrained as desired. Results were very satisfactory. The period of observation lasted five years.

CASE XIX. P. G., a twelve year old girl, had a sudden "caving in" of the right leg while walking. There was no stumbling and no accident. X-ray examination revealed complete intracapsular fracture of the right femoral neck. In view of the very slight injury connected with this complete severance of continuity the possibility of pathological fracture was considered, which was supported by x-ray examination during treatment by Russell traction on October 11, 1936, showing smooth fracture edges. (Fig. 91.) The femoral head showed what was interpreted as a focus of primary compression. In view of the youth of the patient bony union was expected. This seemed to be well under way after seven weeks of treatment, as shown by x-ray examination on November 21, 1936. (Fig. 92.) However, when traction was discontinued after eleven weeks of treatment, the limb fell instantly into external rotation indicating nonunion, which was confirmed by x-ray examination on December 29, 1936. (Fig. 93.) Application

of traction easily restored the reduction previously obtained, but release was immediately followed by falling apart of the fragments. For this reason a plaster of paris spica was applied for eight weeks. After removal of

FIG. 91. Case XIX. Roentgenogram, October 11, 1936; smooth fracture line, marked absorption; primary compression focus in head.

FIG. 92. Case XIX. Roentgenogram November 21, 1936; apparent union.

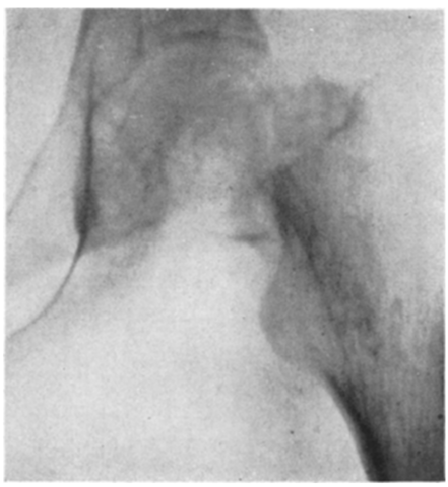


FIG. 93. Case XIX. Roentgenogram after eleven weeks in traction, December 29, 1936; no trace of union, marked external rotation.

the spica, March 8, 1937, there was no sign of union and the thigh was freely telescoping on the side of the pelvis. *X*-ray examination on this day showed the completely rotated head and the externally rotated distal fragment.

(Fig. 94.) Five months of careful immobilization elapsed without bringing any trace of union. The drilling operation was performed April 5, 1937. Owing to the emaciated condition of the child, immobilization was obtained

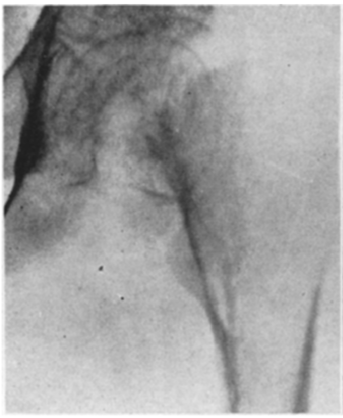


FIG. 94. Case XIX. Roentgenogram five months after accident; eleven weeks traction, eight weeks spica; no trace of union.

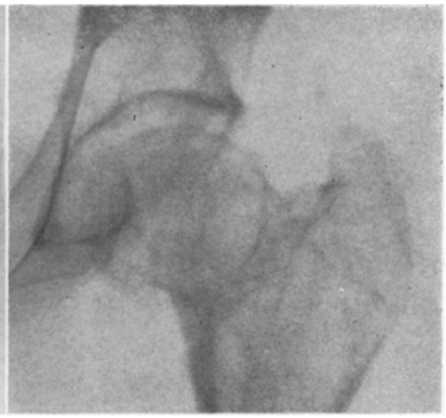


FIG. 95. Case XIX. Roentgenogram two years after drilling, February 24, 1939; firm union, well shaped head.

FIG. 96. Case XX. Roentgenogram July 25, 1936; complete fracture at base of neck, markedly smooth edges.

FIG. 97. Case XX. Roentgenogram July 6, 1938; good union; primary focus in head found.

as best as possible by sand bags. Rapid consolidation ensued and evidences of clinical union appeared after two months. The child was allowed to walk about on crutches four months after drilling. Subsequent clinical and x-ray examinations showed steady improvement. X-ray examination on February 24, 1939, showed complete consolidation of the fracture with fair position

of the fragments and good bone structure. (Fig. 95.) Weight bearing was perfect and painless, and range of motion nearly complete. The child pursues all her activities unhampered and is unaware of any physical shortcomings. The period of observation lasted three and one-quarter years.

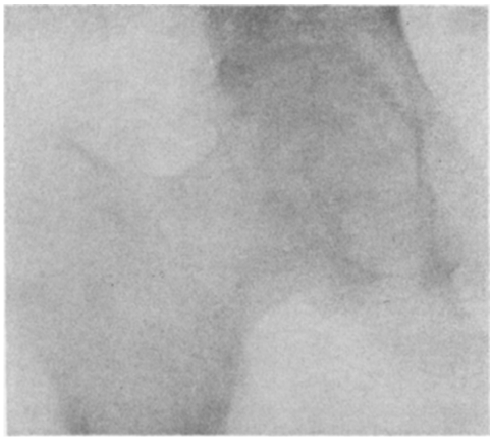


FIG. 98. Case xx. Roentgenogram two and one-half years after drilling, February 20, 1939; firm union.

CASE XX. L. G., a ten year old girl, while stepping on the rungs of a ladder had her right leg "give out" under her. *X*-ray examination, July 25, 1936, showed a complete interruption of continuity at the base of the right femoral neck. This "fracture line" was of such even smoothness that considering the nature of the accident, the diagnosis of a pathological fracture was made. (Fig. 96.) Reduction and drilling were done on July 30, 1936, and a spica applied for eight weeks. Rapid improvement followed. The patient was allowed on crutches after careful exercises in bed gave clinical evidence of union four months after the accident. On repeated *x*-ray examinations, each time in a different position, a search was made for a primary compression focus in the head. This was eventually located on *x*-ray examination made July 6, 1938. (Fig. 97.) A drilling into this focus was recommended but refused. *X*-ray examination on February 20, 1939, showed good consolidation at the fracture site, a well shaped head, satisfactory bone structure and apparently uo untoward development due to the primary compression focus. (Fig. 98.) This may be due to the circumstance that the segment harboring the focus is possibly not subjected to weight bearing. The parents report that the child is well, pursuing all activities and is unaware of any disability. The period of observation covered three and one-half years.

The reported cases of juvenile fractures of the femoral neck indicate in the opinion of the author that one cannot rely implicitly

on the vitality of these young patients to bring about repair. The delay or entire abeyance of repair is due to the presence of compressed necrotic bone at the site of interruption of continuity. The

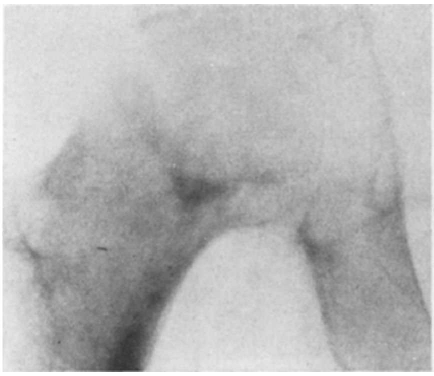


FIG. 99. Case XXI. Left hip; roentgenogram June 23, 1937; wedge shaped compression in femoral head; beginning collapse.

FIG. 100. Case XXI. Right hip of same patient as in Figure 99; roentgenogram June 23, 1937; wedge shape of compression focus very clearly outlined.

demonstrated presence of primary foci in the head corroborates the explanation given by the author as to the mechanism of these injuries. The analysis of these Whitman-type juvenile fractures of the femoral neck may furnish clues to the understanding of the problems of intracapsular hip fractures occurring in adults, as the former represent, as it were, the slow-motion version of the latter.

ARTHRITIS DEFORMANS OF THE HIP

CASE XXI. J. C., a forty-five year old male, was under observation with marked disability of both hips for two and one-half years. Examination revealed in both hips the condition variously called malum senile, arthritis deformans, osteoarthritis, etc. The patient had in his possession *x*-ray pictures taken at the onset of the disability, June 23, 1937. (Figs. 99 and 100.) These indicate with remarkable clearness the wedge shape of the lesion before collapse of the head occurred, indicating that the underlying cause of the disease is compression necrosis of the femoral head. The patient cannot recall any marked single accident, but reports continued hard physical labor over a period of many years prior to the inception of the disability.

INVOLVEMENT OF THE ACETABULUM

CASE XXII. M. F., an eleven year old girl, was under observation at home for pain and limp in the left hip. *X*-ray examination on June 9, 1937,

showed gross disturbance of the upper portion of the acetabulum with the formation of a partially separated sequestrum. The femoral head showed a definite primary compression focus. (Fig. 101.) The position of the two

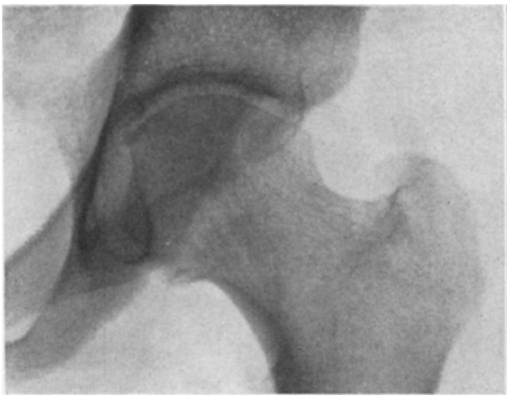


FIG. 101. Case XXII. Roentgenogram June 9, 1937; partly separated sequestrum in acetabulum; compression focus in head.

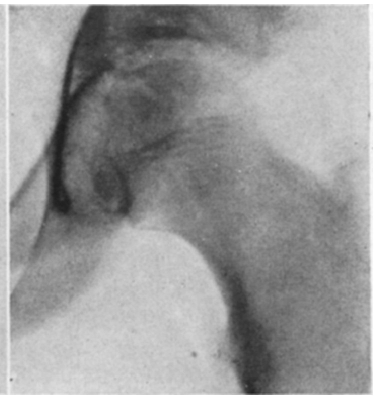


FIG. 102. Case XXII. Roentgenogram two years later, June 2, 1939; focus in acetabulum healed; focus in head still present; beginning spur formation.

foci indicates that the injury to both occurred in marked adduction. Rest in bed and treatment by extension at home, intermittently with restricted activity were carried out. *X*-ray examination two years later, June 2, 1939, showed that the injury in the acetabulum had completely healed insofar as *x*-ray appearance was concerned. The primary compression focus on the head was still discernible with the indication of the formation of a spur. (Fig. 102.) While *x*-ray evidence showed recovery, there are, however, periods of pain and stiffening in the left hip necessitating bed rest and traction.

INVOLVEMENT OF THE SACROILIAC SYNCHONDROSES

CASE XXIII. C. H., a nineteen year old male, had pain in lower part of the back, stiffness, fatigue on slight exertion and inability to walk any but short distances. He also experienced radiating pain along the posterior aspect of both thighs. *X*-ray examination revealed marked bone destruction at both sacroiliac joints. Rest in bed and traction were used but there was little improvement. The author believing, as previously stated, that the condition was due to compression necrosis in the ilium distal to the joints, recommended drilling so as to bring about absorption of necrotic material and possibly fusion of the synchondroses. The drilling operation was performed on February 15, 1933. Drill specimens removed from the ilium revealed massive bone necrosis. (Fig. 104.) *X*-ray examination on March 20,

1933, showed the condition as it was a month after drilling. (Fig. 103.) Temporary improvement occurred but the sympoms, however, returned. Within one year both sacroiliac synchondroses fused firmly, but immedi-

FIG. 103. Case XXIII. Roentgenogram four weeks after drilling, March 20, 1933; marked bone destruction, increased density at both sacroiliac joints, especially in ilium.

ately following, arthritis of the spine (Marie-Struempell type) developed. The patient last seen February 23, 1940, is totally disabled with completely fused spine and other joint involvements.

It cannot be determined, on the one hand, whether fusion of the sacroiliac synchondroses was the result of drilling or of the disease not manifest at that time; on the other hand the possibility, however remote, exists that the drilling released a rush or wave of ossification that progressed out of bounds. Further, it must be considered as possible that there is an introductory stage of the Marie-Struempell arthritis of the spine consisting of compression damages at the sacroiliac synchondroses and along the spine, with the final picture of the disease being the result of the spontaneous ossification of these foci. This case is reported with the recommendation that drilling is not to be performed in these conditions until other cases under observation clarify the various points mentioned.

Another instance in which drilling is not recommended is the involvement of the acetabulum.

It is the impression of the author that where necrotic foci are brought into communication with muscle or connective tissue through drill channels or by their spontaneous breaking open,

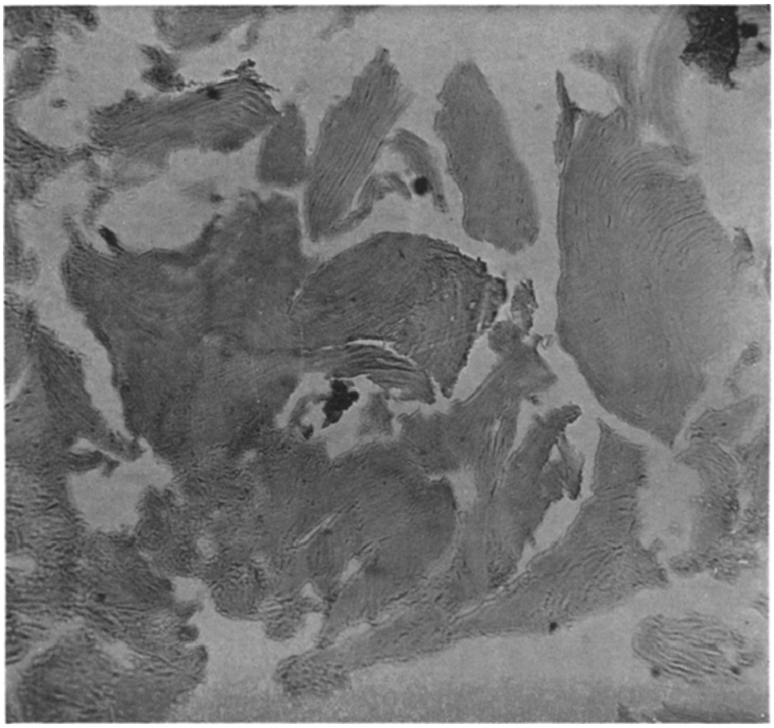


FIG. 104. Case XXIII. Drill specimen; nothing but necrotic bone with empty lacunae, no living marrow elements; marked homogenization and granular breaking up of necrotic bone.

unpredictable and uncontrollable ossification or fibrosis may result. The drilling is safe if it is performed from living bone to dead bone within the boundaries of bone.

Compression and its consequences may develop in cortical bone also. Blaisdell[13] demonstrated that in young individuals at forcible bending, laminae of the cortex slide against and over one another with subsequent injury to the vessels. The author believes that this mechanism is the underlying cause of the so-called spontaneous fractures of shafts of long bones. Mechanical explanation of these occurrences on the basis of Looser's[69] original studies, is convincingly represented by Walter[116] in his later publications.

RÉSUMÉ

The author endeavored to give an outline of the concept of compression of cancellous bone, and to demonstrate that its manifestations occur from earliest childhood to advanced old age in a great variety of clinical pictures.

A method of treatment and its results were demonstrated.

An attempt was made to record the great merits of Palletta, Bell, Ollier and others in laying the foundations for understanding the group of diseases discussed.

The author is greatly indebted to Dr. Leo Mayer for the early and extensive trial accorded the treatment described, that resulted in the mass demonstration of efficacy of drilling in instances of slipped epiphyses. Cases IX and X have been taken from this material. Further, the author wishes to thank the following for the privilege of treating cases, or the use of cases treated, on their respective services: Dr. E. A. Spies (deceased), Case XII; Dr. H. Finkelstein and H. C. Stein, Case XVIII; Dr. W. Galland, Cases II and V; Dr S. Eppstein, Case VII; Dr. Th. J. O'Kane, Case XXII. Thanks are due to Dr. W. Aronson for interpreting histological findings (report of this material under preparation), and to Dr. S. Weitzner for co-operation in *x*-ray examinations.

Besides those mentioned, the author wishes to express his gratitude to Dr. Charles L. Scudder and Dr. John D. Adams for their invaluable advice and support throughout the past nine years, and to Dr. E. G. Brackett for his encouraging criticism.

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